A DISSERTATION ON

"STUDY ON SPECTRUM OF RENAL DISEASES INELDERLY PATIENTSATTENDING A TERTIARY CARE HOSPITAL"

Submitted to

THE DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

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for the Award of the Degree of

M.D. BRANCH -I

GENERAL MEDICINE

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STANLEY MEDICAL COLLEGE

CHENNAI-600 001

CERTIFICATE BY THE INSTITUTION

This is to certify that Dr. M.VEERAPANDIAN, Post - Graduate Student (MAY 2012 TO APRIL 2015) in the Department of General Medicine STANLEY MEDICAL COLLEGE, Chennai- 600 001, has done this dissertation on **"A STUDY ON SPECTRUM OF RENAL DISEASES IN ELDERLY PATIENTS ATTENDING A TERTIARY CARE HOSPITAL**" under my guidance and supervision in partial fulfillmentof the regulations laiddown by the TamilnaduDr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in April 2015.

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DECLARATION

I **Dr. M. VEERAPANDIAN** declare that I carried out this work on **"A STUDY ON SPECTRUM OF RENAL DISEASES IN ELDERLY PATIENTS ATTENDING A TERTIARY CARE HOSPITAL,"**at the Nephrology and Medical wards of Government Stanley Hospital during the period June 2014 to September 2014. I also declare that this bonafideworkor a part of this work was not submitted by me or any other for any award, degree, or diploma to any other university, board either in India or abroad.

This is submitted to The TamilnaduDr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. D. Degree examination in General Medicine.

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CONTENTS

ABSTRACT

STUDY ON SPECTRUM OF RENAL DISEASES IN ELDERLY PATIENTS ATTENDING A TERTIARY CARE HOSPITAL

M.Edwin Fernando , M.Veerapandian

BACKGROUND:

The aim of the study is to study the pattern of renal diseases in elderly subjects and the etiology causing them in patients attending Stanley medical college, Chennai.

MATERIALS AND METHODS: Elderly patients above 65 years (n=75) attending nephrology outpatient department from June 2014 to September 2014 ,with raised urea, creatinine values ,abnormal urinalysis reports and electrolyte imbalance were analysed.

RESULTS : The study population included 48 males and 27 female patients . CKD was seen in 49.33% patients. AKI reported in 36% patients. . Sepsis contributed to large part of AKI (44.4%). dehydration due to gastroenteritis contributed to AKI in 29.62% patients. Other causes are BPH in 14.81% and Carcinoma cervix in 3.7% patients. . Glomerular diseases were seen in 6 patients (8%) . This includes membranous nephropathy (2 patients), acute glomerulonephritis (2 patients) and myeloma kidney seen in 2 patients. Other diseases seen in 6.66% of study population. This includes polycystic kidney seen in 3 patients, simple cysts in 2 patients.

CONCLUSION : Good control of diabetes and hypertension in younger age may prevent the occurrence of CKD as they grow older. Elderly patients should avoid taking over the counter drugs.

KEY WORDS : India ; Elderly patients ; renal diseases; spectrum

INTRODUCTION

One of the most striking changes in the demography of the world has been the increased proportion of elderly individuals in the population, who are considered as the "Geriatric" individuals of age 65 years and above.

The relevance of this to health and social services is that there is exponential increase in disability and mental and physical morbidity in geriatrics.

Ageing can be described from a physiologic standpoint ,as a progressive constriction of the homeostatic reserve of every organ system .This decline referred to as homeostasis is evident by the third decade and is then gradually progressive.

Alterations in kidney function occur with advancement of age .Increased susceptibility to systemic diseases and exposure to multiple drugs makes the elderly people more likely to kidney diseases. This study is about the various pattern of renal diseases among elderly patients and various etiological factors causing them.

AIM OF THE STUDY

The aim and objectives of the study are :

1. TO STUDY THE PATTERNS OF RENAL DISEASES IN ELDERLY PATIENTS

2. TO STUDY THE VARIOUS ETIOLOGIES AFFECTING KIDNEY FUNCTION IN ELDERLY PATIENTS

REVIEW OF LITERATURE

HISTORICAL REVIEW

Glomerular filtration rate :

The modern era of kidney function assessment began with the measurement Of urea. Urea was first isolated from human urine by Rouelle in 1773. In the early 1800s, Fourcroy coined the term " urue", carefully choosing a name that would avoid confusion with "urique or uric acid".

In 1827, Richard Bright observed that urea accumulated in the blood of patients with dropsy and he linked this phenomenon to decreased urine urea concentration, proteinuria and diseased kidneys. One year later, Wohler synthesized urea from ammonium cyanate; in so doing he helped discredit the doctrine of vitalism, which was then prevalent.

In 1842, Dumas and Cahours demonstrated that urea was a product of dietary protein catabolism and in 1903,strauss introduced blood urea level as a diagnostic test for kidney disease.

Homer Smith credited Ambard and Weil with one of the first attempts to measure kidney function “dynamic” test in 1912 . These researchers characterized kidney function (K) as blood urea concentration (B) divided by the product of the square root of the urea excretion(D) times the square root of urine urea concentration (U), as follows:

$$K = B(\sqrt{D} \times \sqrt{U})$$

In 1926,Rehberg used exogenous creatinine to measure renal clearance as a measure of glomerular filtration. In 1928, Addis described kidney function as a urea excretion ratio, or the quantity of urea excreted divided by the concentration in blood. Around the same time, the concept of urea clearance as a measure of kidney function was described in detail by Moeller, McIntosh and Vanslyke.

Embryology of kidney:

The embryological development of kidney has three stages: the pronephros, mesonephros, and metanephros. The pronephros is the early stage and metanephros is most developed. The metanephros develops into adult kidney.

During the 5th week of pregnancy, the mesonephric duct forms an outpouching, known as ureteric bud, near the attachment to the cloaca. This bud, also known as the metanephrogenic diverticulum, develops backwards and towards the head of the embryo.

The elongated stalk of the ureteric bud, known as metanephric duct which develops into the ureter. The cranial end of the bud undergoes progressive division to form the collecting duct system of the kidney. It also forms the renal pelvis, major and minor calyces.

The metanephrogenic blastema develops from the intermediate mesoderm which is in contact with branching ureteric bud. The meta nephrogenic blastema differentiates into the renal tubules.

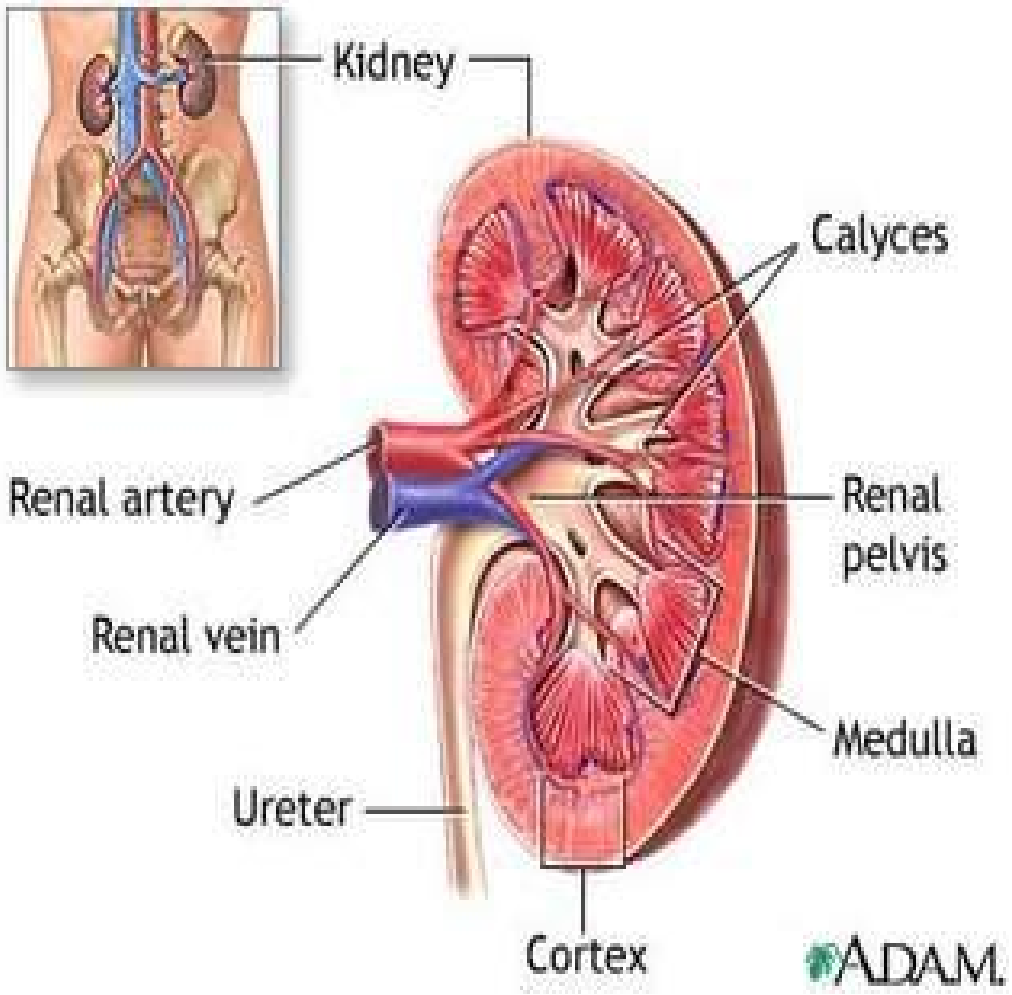
The renal tubules develop and join with connecting tubules of the collecting duct system, forming a continuous tube for flow of urine from the renal tubule to the collecting duct. At the same time, precursors

of vascular endothelial cells develop to assume their position at the tips of the renal tubules. These cells develop into the cells of the future glomerulus.

In humans, all of the divisions of the ureteric bud and the nephronic units are formed by 32 - 36 weeks of pregnancy. These structures are not yet fully developed and will continue to mature after birth. Once matured, each human kidney will have about 1 million nephrons.

ANATOMY OF KIDNEYS:

The kidneys are a paired organs situated one on either side of inferior vena cava and aorta . They are retroperitoneal organs. They extend between 12th thoracic and third lumbar Vertebrae. The right kidney is slightly lower than left. They weigh about 150g in males and 135g in females. They have a length about 10-12 cm, width about 5-7cm , and thickness about 2-3 cm



RELATIONS:

Superiorly, suprarenal glands lie adjacent to the upper pole of each kidney. On the right side, the second part of the duodenum is related to the inner aspect of the kidney

On the left side, the greater curvature of the stomach is related to superomedial aspect of the kidney, and the tail of the pancreas may extend to overlie the renal hilum

The spleen is located anterior to the upper pole and is connected by the splenorenal ligaments. Posteriorly diaphragm is related to upper end of each kidney. The kidney lies over psoas muscle medially and quadratus lumborum laterally.

BLOOD SUPPLY:

The kidneys receive 20% of cardiac output. They are supplied by paired renal arteries. The renal artery divides into 5 segmental arteries which branches into interlobar arteries. The interlobar arteries gives rise to arcuate arteries which inturn leads to interlobular arteries. The interlobular arteries become afferent arteriole then peritubular capillaries and finally efferent arterioles.

Venous drainage is mainly by renal vein which lies anterior to the artery at the hilum. The left suprarenal and gonadal vein drains into left renal vein while on the right side they drain directly into IVC

Lymphatic drainage follows the venous drainage. The left kidney drains into Lt.lateral aortic lymph nodes while right kidney drains to Rt.caval nodes.

Nerve supply:

The kidney receives autonomic supply via the parasympathetic and sympathetic portions of nervous system. The preganglionic sympathetic nervous innervation to the kidneys arises from the T8-L1 segments of spinal cord.

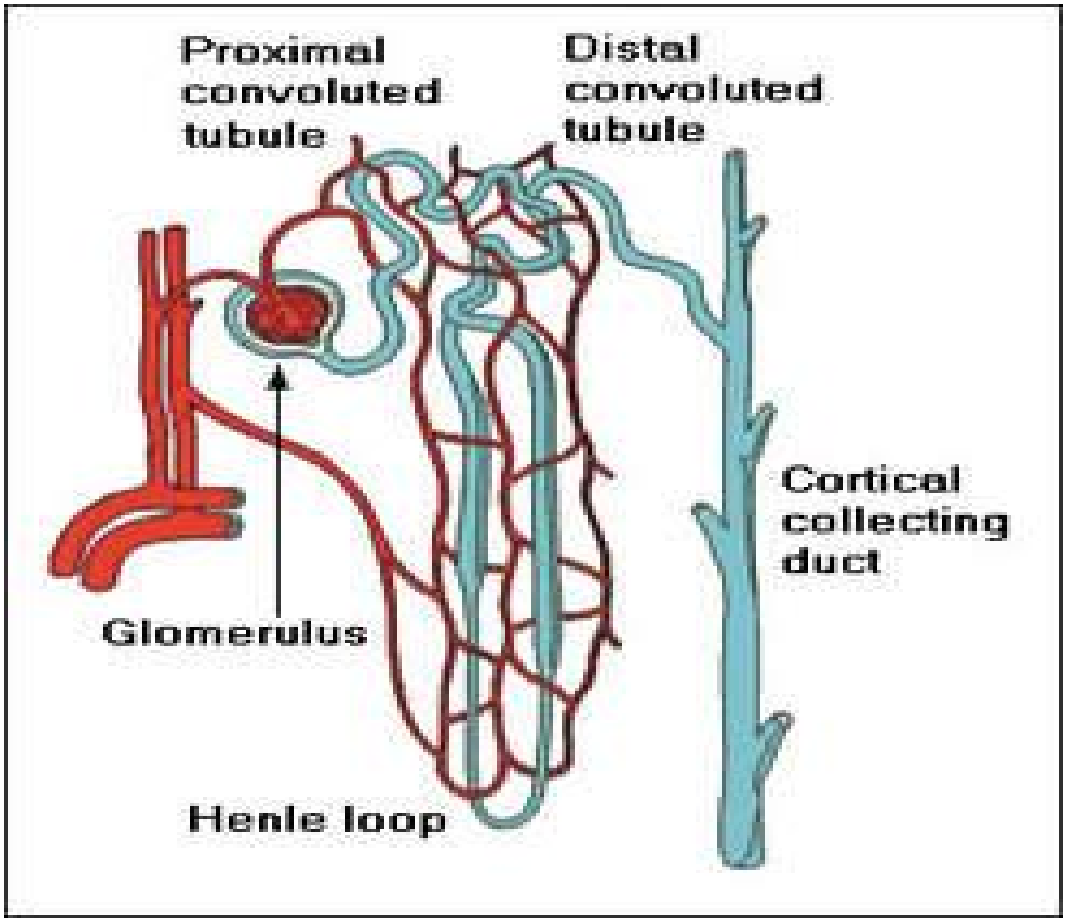
Activation of sympathetic system causes vasoconstriction of the renal vessels. Parasympathetic innervation arises from the vagus nerve, and causes vasodilation when stimulated.

Microscopic structure:

Each kidney contains about 1 million nephrons.

A nephron is mainly made up of glomerulus which drain into tubules.

Both human kidney contain about 1.8 million glomeruli. Each glomerulus consists of an afferent arteriole, efferent arteriole and Bowman's capsule.

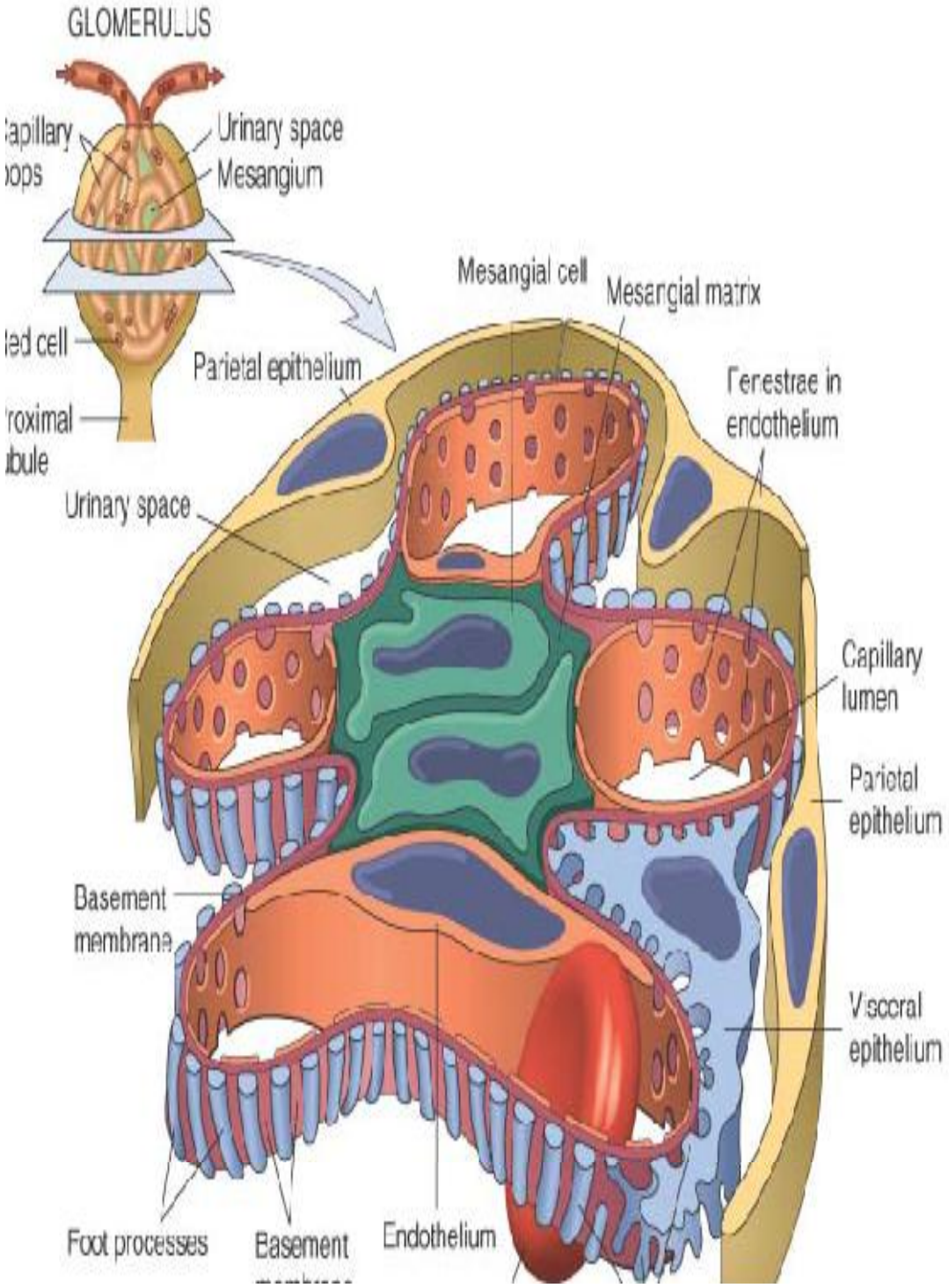


The afferent arteriole derived from the terminal branches of renal artery divides into tuft of capillaries embedded in mesangium and reunite to form an efferent arteriole which drains into peritubular capillaries.

Glomerular capillary wall is made up of, from inside to outside, endothelium, basement membrane and visceral epithelium. There are two types of epithelial cells, one is visceral epithelium which covers the glomerular capillaries and other is the parietal epithelium that coats the Bowmans's membrane from inside.

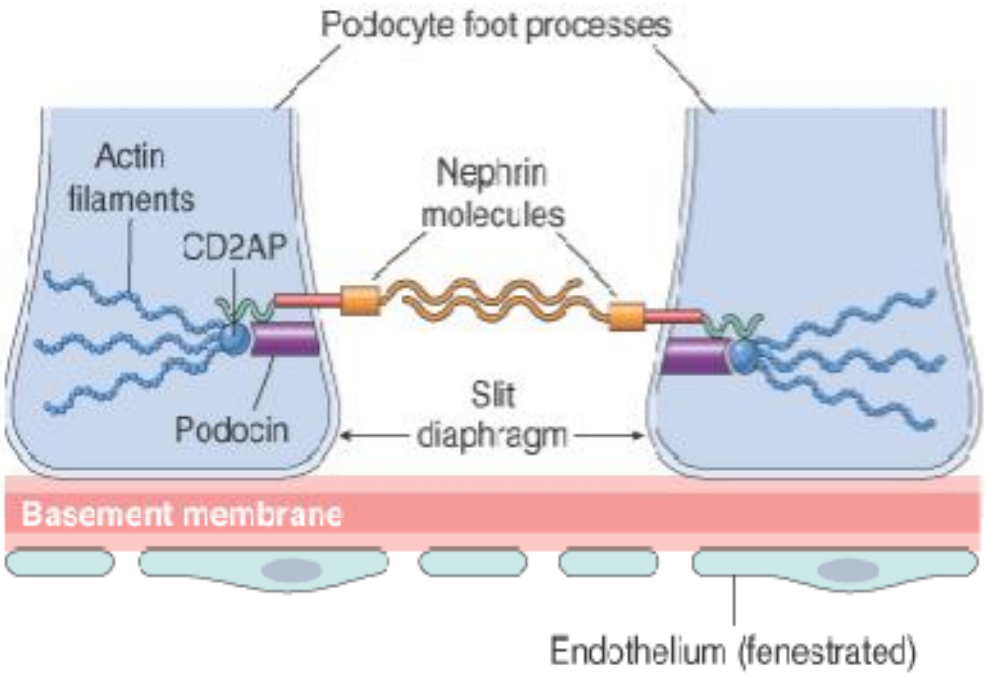
The visceral epithelium is a specialized cell which has a lot of foot process called podocytes which covers the capillaries. The mesangium contains a group of cells which are similar to pericytes of blood vessels and extracellular matrix.

Mesangium secretes many biologically active substances. The endothelium has many fenestrations made by foot processes. The distance between two foot process is about 20-30 nm which are called as "filtration slits". It allows substances of size less than 70 kilodaltons only and it is impermeable to proteins which are larger material.


GLOMERULUS
Capillary loops
Urinary space
Mesangium
Mesangial cell
Mesangial matrix
Parietal epithelium
Fenestrae in endothelium
Urinary space
Capillary lumen
Parietal epithelium
Basement membrane
Visceral epithelium
Foot processes
Endothelium

Glomerular basement membrane:

Renal glomerular basement membrane is a unique structure which has selective permeability. It has three layers a thick outer electron dense layer lamina densa in the middle and electron lucent lamina rara interna inside and lamina rara externa outside. The GBM is mainly made up of collagen type IV and other substances like fibronectin, laminin, entactin, proteoglycans and glycoproteins. The foot processes are bridged by nephrin molecule which get attached to Podocinin cell membrane connected to actin filaments inside the cell.



Mutations in these proteins are responsible for many diseases.

Functions of Glomerulus:

The major function of glomerulus is filtration. The GBM is a selectively permeable membrane with charge selectivity and size selectivity. The GBM is made up of negatively charged proteoglycans which repels the negative charged albumin like substances. The size selectivity is important that electrolytes, water, inulin are freely filtered whereas albumin and myoglobin are not filtered. Many of the water soluble drugs and normal metabolites of body are filtered only through kidneys.

The mesangium is a specialized connective tissue which surrounds glomerular capillaries and fills the spaces between endothelial surfaces that are not invested by podocytes Mesangium is made up of pericytes, phagocytes, Juxtaglomerular cells and some paracrine cells which has number of functions. It secretes prostaglandins, nitric oxide and proinflammatory cytokines.

The glomerulus is connected to renal tubule. The renal tubule is divided into proximal tubule, ascending and descending loops of henle, distal convoluted tubule.

The human proximal convoluted tubule is about 15 mm long and 55 um in diameter. Its wall is made up of a single layer of cells that are closely connected with one another and are united by apical tight junctions. The luminal side of the cells have a brush border due to the presence of microvilli. The proximal tubule drains into the straight portion (pars recta), which forms the first part of the Henle's loop. The proximal tubule terminates in the thin segment of the descending limb of the loop of Henle.

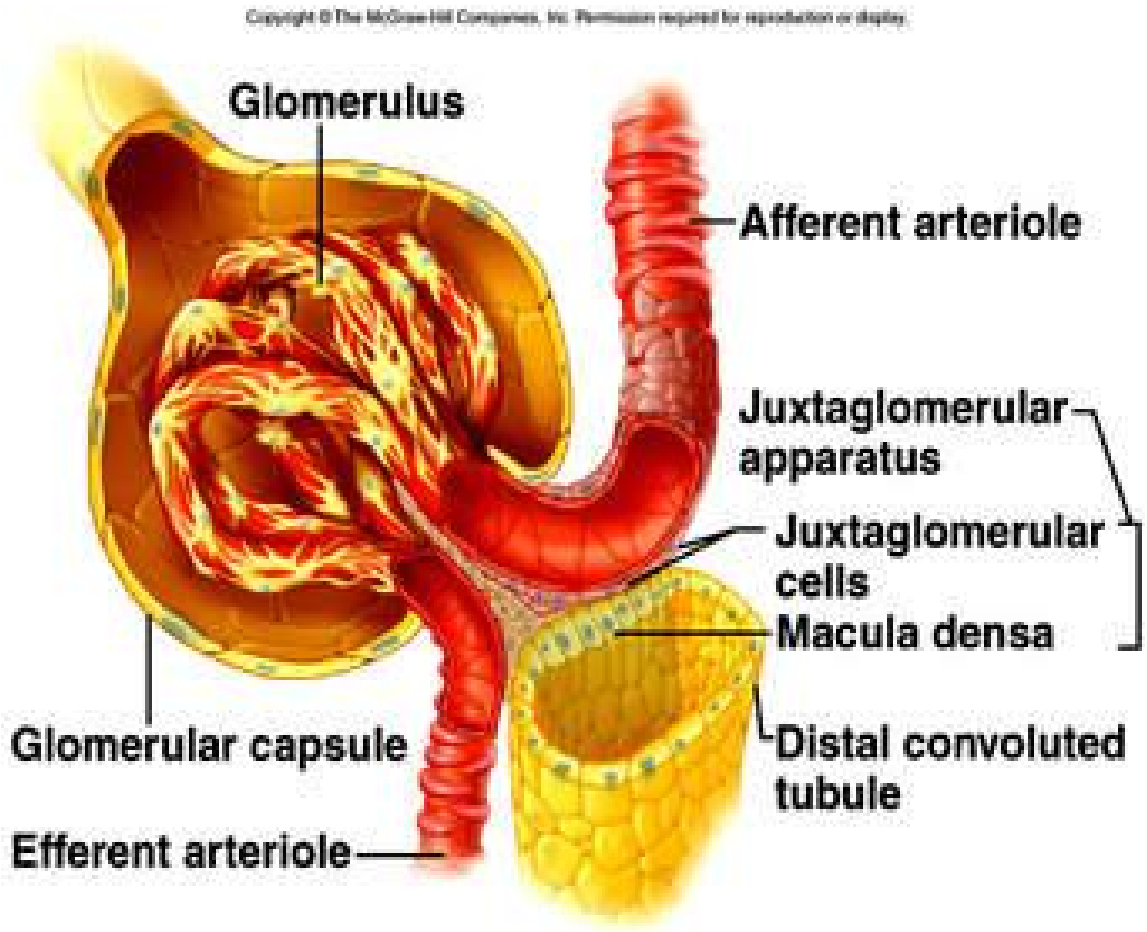
The nephrons with glomeruli in the outer portions of the renal cortex have short loops of Henle(cortical nephrons), whereas those with glomeruli in the juxtamedullary region of the cortex (juxtamedullary nephrons) have long loops.

The total length of the thin segment of the loop varies from 2 to 14 mm. It ends in the thick segment of the ascending limb, which is about 12 mm in length. The cells of the thick ascending limb are cuboid.

The thick ascending limb of the loop of Henle passes close to the glomerulus of the nephron from which the tubule arose. The lining epithelium of the afferent arterioles contain the juxtaglomerular cells.

At this junction, epithelium of the proximal tubule is modified to form the macula densa.

The maculadensa, juxtaglomerular cells and the lacis cells near them are known collectively as the juxtaglomerular apparatus.



The distal convoluted tubule is about 5 mm long. It has few microvilli but no distinct brush border. The distal tubules join to form collecting ducts that measures 20 mm in length and pass through renal cortex and medulla to drain into the pelvis of the kidney at the apexes of the pyramids.

The epithelium of the collecting ducts is made up of principal cells (P cells) and intercalated cells (I cells). The P cells, are relatively tall and have few organelles. They are involved in Na+ reabsorption and vasopressin-stimulated water reabsorption.

The I cells have more microvilli, cytoplasmic vesicles, and mitochondria. They are involved in acid secretion and bicarbonate transport.

The collecting duct system includes the connecting tubule and collecting duct. The collecting ducts may be subdivided into cortical and medullary ducts. The collecting ducts fuse and open finally as papillary ducts into the renal pelvis.

Cortical nephrons perform most of glomerular filtration because their afferent arterioles are larger than efferent arteriole. The juxta

medullary nephrons create a hyperosmolar gradient for concentrating urine.

RENAL BLOOD FLOW:

Renal blood flow can be measured can be determined by applying the Fick principle to the kidney—ie, by measuring the amount of a given substance taken up per unit of time and dividing this value by the arteriovenous difference for the substance across the kidney. Since the kidney filters plasma, the renal plasma flow equals the concentration of a substance excreted per unit of time divided by the renal AV difference.

Any excreted substance can be used if its concentration in arterial and renal venous plasma can be measured and if it is not metabolized, stored, or produced by the kidney and does not itself affect blood flow.

Renal plasma flow can be measured by infusing p-aminohippuric acid (PAH) and determining its urine and plasma concentrations. PAH is filtered by the glomeruli and secreted by the tubular cells, so that its extraction ratio (arterial concentration minus renal venous concentration divided by arterial concentration) is high.

The value obtained should be called the effective renal plasma flow (ERPF) . In humans, ERPF averages about 625 mL/min

Glomerular filtration rate:

Filtration is determined mainly by size of the molecule and shape of the solute and also by its charge. Substances with size exceeding 4 nm are not filtered. The negatively charged GBM also restricts the filtration of proteins like albumin.

Autoregulation of GFR is the result of 3 factors : myogenic reflex, Tubulo Glomerular feedback and angiotensin II mediated vasoconstriction of efferent arteriole.

Characteristics of an ideal exogenous or endogenous marker:

1.safe and convenient

2.readily diffusible in extracellular spaces.

3. freely filterable

4. No tubular reabsorption

5.No tubular secretion

6. No extrarenal elimination or degradation

7. No compounds interfere

8. inexpensive

9.No influence on GFR.

Glomerular filteration cannot be measured directly. If a substance in stable concentration in the plasma is physiologically inert, freely filtered at the glomerulus and neither secreted, reabsorbed nor metabolized by the kidney ,the amount of that substance filtered at the glomerulus is equal to the amount excreted in the urine. Inulin has each of the above properties and has long been considered an ideal substance to estimate GFR.

The amount of inulin filtered at the glomerulus equals the GFR multiplied by the plasma inulin concentration : GFR × P_in. The amount of excreted inulin equals the urine inulin concentration (U_in) multiplied by the urine flow rate (V).

Since filtered inulin= excreted inulin:

$$GFR \times P_in = U_in \times V$$

$$GFR = (U_in \times V) / P_in$$

The factors regulating filtration across the glomerular capillaries are the same as those governing filtration across all other capillaries,ie, the size of the capillary bed, the permeability of the capillaries, and the hydrostatic and osmotic pressure gradients across the capillary wall. For each nephron:

$$GFR=Kf[\ (PGc - Pt) - (\pi Gc - \pi t)]$$

Kf, the glomerular ultrafiltration coefficient, the product of its permeability and the effective filtration surface area. PGC is the mean hydrostatic pressure in the glomerular capillaries, Pt the mean hydrostatic pressure in the tubule, πGC the osmotic pressure of the plasma in the glomerular capillaries, and πt the osmotic pressure of the filtrate in the tubule.

The amount of protein in the urine is normally less than 100 mg/d, and most of this is not filtered but comes from shed tubular cells. The presence of significant amounts of albumin in the urine is called albuminuria. In nephritis, the negative charges in the glomerular wall are dissipated, and albuminuria can occur for this reason without an increase in the size of the "pores" in the membrane.

Na+ Reabsorption

The reabsorption of sodium and Chloride has a important role in electrolyte and water metabolism. In addition, Na+ transport is coupled to the transport of H+, other electrolytes, glucose, amino acids, organic acids, phosphate, and other substances across the tubule walls.

Sodium is transported by means of active transport out of all parts of renal tubule except thin portions of the loop of Henle. Na+ is pumped into the interstitium by Na+-K+ ATPase. It extrudes three Na+ in exchange for two K+ that are pumped into the cell. Much of the Na+ is actively transported into the lateral intercellular spaces .

Glucose, amino acids, and bicarbonate are reabsorbed along with sodium in the early part of the proximal tubule . Glucose is removed by means of secondary active transport. It is filtered at a rate of approximately 100mg/min . Most of the glucose is reabsorbed. Only a few milligrams appear in the urine per 24 hours.

The amount reabsorbed is proportionate to the amount filtered and hence to the plasma glucose level (PG) multiplied by the GFR up to the transport maximum (TmG); but when the TmG is exceeded, the amount of glucose in the urine rises . The TmG is about 375 mg/min in men and 300 mg/min in women.

The plasma level at which the glucose first appears in the urine in more than the normal minute amounts is known as renal threshold. The renal threshold is normally about 300 mg/dL. However, the actual renal threshold is corresponds to a venous level of about 180 mg/dL. The difference between the ideal and actual threshold is called splay.

Glucose transport mechanism:

In the early part of the PCT , sodium and glucose are cotransported from the tubular lumen by SGLT 2, Na+ is pumped out of the tubular cells by Na+-K+ ATPase in the basolateral membranes, and glucose is transported to the interstitium by GLUT 2. Other substances likeaminoacids , lactate, inorganic phosphate ,H^+ and $cl^-$are also transported along with sodium

Water excretion:

The total amount of fluid filtered through the glomerulus is about 180 litres /day. But daily urine volume averages about 1 litre .At the end of the proximal tubule,70% of the filtered solute and water is removed.

The descending limb of Henle's loop is permeable to water, but the ascending limb is not permeable. Distal tubule is also impermeable to water.

The antidiuretic hormone increases the permeability of the collecting ducts to water. The key to the action of vasopressin on the collecting ducts is aquaporin-2. Unlike the other aquaporins, this aquaporin is stored in vesicles in the cytoplasm of principal cells. Vasopressin causes rapid insertion of these vesicles into the apical membrane of cells**.**

Mechanisms of progressive kidney disease:

1. glomerular hyperfiltration and hypertension.
2. proteinuria
3. cytokine bath
4. nephritogenic T lymphocyte activation
5. epithelial mesenchymal transition
6. fibrosis

With the loss of functioning nephrons, glomerulotubular balance is maintained, by which the residual tubules adjust to increases in glomerular filtration with necessary alterations in reabsorption or excretion of filtered water and solutes in order to maintain homeostasis.

TUBULAR FUNCTION IN CHRONIC RENAL FAILURE:

1. The tubules lose the capacity to excrete sodium leading to intravascular volume expansion, edema and hypertension

2. Patients with progressive renal injury gradually lose the capacity either to dilute or concentrate their urine, and urine osmolality becomes fixed about 350mOsm/L

3. Potassium excretion is impaired leading to hyperkalemia

4. With loss of renal function, there is loss of H+ pumps in the intercalated cells of collecting duct and reduction in ammoniagenesis leading to non delta acidosis

5. Calcium and phosphate homeostasis is impaired leading to hypocalcemia, increased PTH secretion and hyperphosphatemia, bone demineralization leading to secondary hyperparathyroidism

The elderly population is the rapidly growing age group in the World, and they constitute the largest group with CKD. The GFR begins to decline at age 40 and drops by 8 ml/min/1.73 m². Elderly individuals lose 30% of kidney size by 8th decade and renal mass decreases to 300 g by the age of 90 years.

Histologic changes in the aging kidney:

Glomerulus - basement membrane becomes thickened, increase mesangial matrix, focal and global sclerosis, hypertrophy.

Podocytes - Fusion, detachment, vacuoles. Tubules undergo atrophy, tubular cast, monocytes infiltrates, fibrosis of interstitium Atrophy of afferent and efferent arterioles may be seen. The vessels sometimes develop hyalinosis and aglomerulus vessels are formed

Functional changes in the aging kidney:

- After 40yrs renal blood flow decreases by 10% per year.
- Reduction in GFR by 0.87 ml/min per year.
- Accelerated Renal vascular resistance
- Diluting capacity is reduced
- Reduction in concentrating capacity

- Renal reserve is maintained

Methods of determining GFR:

Ideal filtration marker: inulin clearance is the Gold standard for GFR assessment but is difficult to use in routine practice Other exogenous markers : Iohexol ,51Cr EDTA; 125 I-iothalamate, 99mTc-DTPA.

Endogenous markers:

Endogenous substances, such as serum creatinine or serum cystatin C can be used to estimate GFR.

Limitations of creatinine:

1.Creatinine generation :

Creatinine generation is proportional to the muscle mass. As a result creatinine generation is higher in male than in women, in younger than in older individuals and in blacks than in whites. Creatinine generation is also affected by meat intake because the process of cooking converts a variable portion of creatine to creatinine. Creatinine production is also affected by low protein diet.

2. Creatininefiltration:

As age advances, excretion decreases resulting in underestimation of GFR.

3. Creatinine Secretion:

(i) because of tubular secretion, tends to overestimate GFR by about 10%. (ii) drugs like cimetidine, trimethoprim inhibit secretion

4. Creatinineassay :

Large differences between laboratories in calibration of the creatinine assays may lead to differences in interpretation of values.

5. Increase in extrarenal elimination with decreasing GFR (degradation of creatinine by bacterial overgrowth in small bowel)

CYSTATIN C:

1. It is used as an substitute to serum creatinine or its estimation equations in elderly people susceptible for muscle mass variation and hence, creatinine production

2. It used as a substitute for serum creatininein identifying old people with initial decrease in GFR.

3. Serum cystatin C–based GFR equations were nearly comparable to MDRD estimates and an equation combining it with serum creatinine, age, sex and race yielded the best measurement of GFR

DISADVANTAGES :

1. There is no definite normal range and , method of estimation are not standardized for clinical use.

2. The test is not commonly used and availability is rare. It is very expensive compared to serum creatinine

3. Excreted more extra renally in renal failure.

The two most frequently used equations for evaluation GFR are serum creatinine based: Cockcroft-Gault (CG) and the Modification of Diet in Renal Disease (MDRD) equations.

CG EQUATION:

This is one of the most frequently used equations, among aged people, although it was initially derived from younger individuals.

The CG equation is as follows:

$$\text{Cr.cl} = \frac{[140 - age\ in\ yr] - weight\ in\ kg}{\text{s.creatinine(mg/dl)} \times 72}$$

The above equation should be multiplied by 0.85 for female.

MDRD equation is as follows:

$$\textbf{GFR} = 1.86 \times (\textbf{Serum creatinine})^{-1.154} \times (\textbf{age})^{-0.203}$$

Multiply by 0.742 if patient is female

Multiply by 1.212 if patient is black

Serum Creatinine and Cystatin C based equation:

Estimated GFR = 177.6 × S.creatinine× 0.65×Cystatin $c^{-0.57}$× $Age^{-.20}$ × (0.82) if female, ×(1.11) if black.

Limitations of CG and MDRD equations

1. Renal or extrarenal conditions influencing plasma level of creatinine.

2. Factors modifying creatinine measurement -Ketones, glucose, drugs such as cephalosporins altering the assay

3. Differences in muscle mass or food intake - Extremes of body mass, only vegetarian diet or protein-rich food or persons who are amputated.

4. Standardization error

Presently, MDRD is the widely used method to measure GFR in older individuals.

CKD was defined by the reduction of glomerular filtration rate (GFR) to less than 60 mL/min/1.73 m²and/or presence of kidney damage, such as proteinuria (albuminuria >30 mg/g of creatinine), glomerular-based or tubular-based hematuria, or abnormal renal imaging and pathologic abnormalities of 3 months' duration or longer, irrespective of the cause.

In 2002, the national kidney foundation kidney disease outcomes quality initiative **(KDOQI)** published guidelines to standardize definition of CKD.

	eGFR		**Markers of kidney damage**
Stage one	>90	Normal to high	Present
Stage two	60 -89	Slight	Present
Stage three	30-59	Moderate	Not required
Stage four	15-29	Severe	Not required
Stage five	< 15	Kidney failure	Not required

Kidney damage is referred to as deviations or markers of Injury, including abnormalities in blood or urine biochemistries or radiological studies.

This guidelines has many limitations.

As the eGFR formulas contain age, older individuals are more likely to be classified as stage 3 CKD. Many of these individuals have normal serum creatinine, no albuminuria, and values that are in the range 50 to 60 mL/min/1.73 m^2. Such values could represent misclassification of CKD.

These findings have led to an updated classification of stage of CKD by **the Kidney Disease Improving Global Outcomes (KDIGO**), published in January 2013. Similar to KDOQI, CKD in KDIGO is defined as "abnormalities of kidney structure or function present for >3 months with implications for health." If the abnormalities are present for longer than 3 months, CKD is confirmed. If it is present for less than 3 months, it is not confirmed. The KDIGO classification changes the 1-stage classification to a classification based on cause of kidney disease, GFR category, and albuminuria category.

For the causes of CKD, one first defines whether the kidney disease is related to systemic disease (eg, diabetes or collagen vascular disease), and then to the presumed or observed location of the anatomic or pathologic changes in the kidney (eg, glomerular, tubulointerstitial, vascular [including hypertension], cystic, and congenital). For the GFR stages G1 and G2 the presence of other markers of kidney damage is required to be considered CKD.

With the joint consideration of albuminuria and GFR, individuals can be classified for risk of progression.

KDIGO classification of CKD:

GFR STAGES	**GFR (ml/min/1.73 m²)**
G 1	>90
G2	60-89
G 3a	45-59
G 3b	30-44
G 4	15 -29
G 5	<15

Albuminuria stages	AER or ACR (mg/24h or mg/g)
A 1	<30
A 2	30-299
A 3	>300

AER- Albumin excretion rate or albumin creatinine ratio

One approach that may help define who has CKD is to use cystatin C as a confirmation measure. Cystatin C is an endogenous protein like, but is definitely produced by all nucleated cells, freely filtered, reabsorbed, and catabolized by the proximal tubules. Most researches have shown that serum cystatin C levels associate better with GFR than does serum creatinine alone, Cystatin C is not dependent on muscle mass and its levels may be influenced by thyroid disease, steroid use, and inflammation.

KDIGO suggested that in individuals with stage 3a GFR but no other markers of kidney damage, a cystatin-based eGFR be calculated. If the cystatin C eGFR is greater than 60, CKD is not confirmed.

RISK FACTORS FOR CKD :

The most common risk factors for CKD are diabetes and hypertension. Longer duration and poorer control of either of these conditions predispose greater risk for CKD. Other risk factors include obesity, smoking, hyperuricemia, dyslipidemia, heart diseases, and a family history of kidney disease. Certain conditions such as heart failure or cancers that are more prevalent in elders predispose individuals at risk for CKD.

Hypertension :

Hypertension, particularly systolic hypertension, is one of the strongest risk factors for CKD. The target BP in individuals with CKD is controversial. The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), the American Diabetes Association, and the prior KDOQI guidelines recommended a target BP of <130/80 mm Hg in individuals with Chronic kidney disease.

As part of the CKD guidelines, the KDIGO also made a BP recommendation to slow progression of kidney disease, recommending a target BP of 140/90 mm Hg or less if albuminuria is less than 30 mg/d and a BP of 130/80 mm Hg or less if albuminuria is 30 mg/dl or greater.

Diabetes mellitus:

The most common disease that will cause proteinuric kidney disease in the elderly is diabetes.

Functional changes in diabetic nephropathy :

Three cardinal functional changes characterize the natural history of diabetic nephropathy. They are,

1) Changes in giomerular filtration rate

2) Proteinuria and albuminuria

3) Changes in arterial pressure.

A study of these functional changes will be made easy if one understands the five stages of diabetic nephropathy.

Stages of diabetic Nephropathy

1. Stage of hyperfunction and hypertrophy:

This stage is characterized by large kidneys and glomerular hyper filtration and hypertrophy. The basement membrane mesangium is normal. The GFR IS > 150 ml/min with normal blood pressure. The urinary albumin excretion may be increased.

2 . Silent stage:

In this stage the blood pressure and urinary albumin excretion are normal. But structural changes like increased basement membrane thickening and mesangial expansion may be present. This situation may last for years.

3. Stage of incipient diabetic nephropathy :

The patient in this stage are at risk to develop overt nephropathy if left untreated. There is persistent microalbuminuria and hypertension.

4. Stage of overt diabetic nephropathy :

This stage is characterized by proteinuria, hypertension and a fall in GFR.

5. Stage of ESRD

This stage is characterized by uremia with generalized nephron closure and very low GFR.

Pathology of diabetic Nephropathy:

The key change in diabetic glomerulopathy is augmentation of extracellular material. The earliest morphologic abnormality in diabetic nephropathy is the thickening of the glomerular basement membrane (GBM) and expansion of the mesangium due to accumulation of extracellular matrix.

The nodular lesion described by kimmelsteil and Wilson has been considered virtually specific for diabetes. The nodules are well demarcated, hard eosinophilic and periodic-Acid Schiff (PAS) positive located in the central region of peripheral glomerular lobules. The nodules are seen in the presence of diffuse lesions and this reflects their appearance only after a long period of disease.

The diffuse glomerular lesion comprises increase in mesangial matrix extending to involve capillary loops. In more severe cases the capillary wall thickening and mesangial expansion lead to capillary narrowing and eventually to complete hyalinisation. In this advanced state, periglomerular fibrosis is present. The exudative lesions are highly eosinophilic, rounded homogenous structures seen in the capsular space overlying a capillary loop(fibrin cap) or lying on the inside of bowman's capsules (capsular drop).

The glomeruli and kidneys are typically normal or increased in size initially thus distinguishing diabetic nephropathy from most other forms of chronic renal insufficiency, wherein renal size is reduced (except renal amyloidosis and polycystic kidney disease).

Immunofluorescence microscopy may reveal deposition of immunoglobulin G along the GBM in a linear pattern but this is not diagnostic. immune deposits are not observed. The renal vasculature typically displays evidence of atherosclerosis usually due to concomitant hyperlipidemia and hypertensive arteriosclerosis.

The severity of diabetic glomerulopathy is estimated by the thickness of the peripheral basement membrane and mesangium and matrix expressed as a fraction of appropriate spaces (eg. Volume fraction of mesangium/glomerulus, matrix/mesangium or matrix/ glomerulus).

Three major histologic changes occur in the glomeruli of persons with diabetic nephropathy. First, mesangial expansion is directly induced by hyperglycemia via increased matrix production or glycosylation of matrix proteins. Second, GBM thickening occurs. Third, glomerular sclerosis is caused by intraglomerular hypertension.

CLINICAL COURSE :

a. Early phase

The increase in GFR and renal plasma flow is accompanied by increase in the kidney size by about 20%. In the silent phase there is no clinically evident proteinuria but a sensitive radioimmuno assay for urinary albumin shows supranormal excretory rates in a proportion of the population. This subclinical albumin excretion is termed as microalbuminuria. In normal individuals it is 1.5 to 20 µg/min and these levels aretermedmicroalbuminuria. The term persistent or clinical albuminuria is used when the AER is more than 200 µg/min. The prevalence of microalbuminuria ranges from 5-37% in type 1 DM and around 47 % in type 2 DM subjects.

Late phase:

This phase is also called clinical nephropathy. The appearance of persistent proteinuria(300 mg/day) signals the onset of clinical nephropathy. There is a gradual decline in GFR to end stage renal failure.

Serum creatinineis found to rise sooner in proteinuric patients with high blood pressure.

In established nephropathy arterial blood pressure is elevated but in some the BP is in the normal range. Fluid retention is common with the advent of clinical proteinuria. A diabetic can have primary or any secondary form of hypertension resulting from diabetic nephropathy, described as "diabetic hypertension". It is volume dependent, low renin, low aldosterone hypertension that responds well to diuretics and fluid restriction.

The data suggest that at least for overtly proteinuricdiabetic kidney disease (urine albumin/creatinine ratio >300 mg/g), blockade of the renin- angiotensin system slows kidney disease.

The KDIGO recommended ACEIs or ARBs for all individuals with diabetes and an albuminuria level of 30 mg/d or higher, and for nondiabetics if at least 300mg/d.

In many randomized studies tighter glucose control was associated with less frequent development or progression of microalbuminuria. However, none of these studies showed an association between tight glucose control and a lesser decline in GFR.

As tight control is not associated with improved outcomes and the risk of hypoglycemia is higher in those with CKD, the recent KDIGO CKD guideline has proposed different hemoglobin A1c targets in those with diabetes and CKD.

The recommended hemoglobin A1c level is approximately 7%. Individuals at increased risk for hypoglycaemia should not be treated to a Hb A1c level lower than 7%.

Acute Kidney Injury:

Acute kidney injury (AKI) is increasingly recognized as a risk factor for both the development of CKD and its progression in the older adult population.

Furthermore, CKD is recognized as a risk factor for the development of AKI relating to decreased renal reserve. Older individuals are at increased risk for AKI, owing to aging-related decreased renal reserve as well as an increased use of medications and procedures that increase the risk of AKI.

NONRENAL-RELATED OUTCOMES

1. Cardiovascular :

Renal disease has been shown to be an important factor influencing heart disease and patients with CKD should be considered a “highrisk” individuals for heart disease.

Common risk factors are :

Old age, Male sex,White individuals, elevated BP , increased LDL-cholesterol, reduced HDL level, type 2 DM /impaired glucose tolerance, smoking, sedentary life style, menopause, stress and depression, family history of heart disease.

CKD related risk factors :

Reduced kidney function, Renin-angiotensin-system activity, proteinuria, volume overload, hyperphosphatemia, Higher PTH level, Anemia, Malnourishment, Inflammatory stimuli, Oxidative stress, Infection, elevated homocysteine levels, hypercoagulable factors.

ST-elevation myocardial infarction is rare in patients with chronic kidney disease. Patients with CKD present rarely with typical symptoms like arm, shoulder, or chest pain or with increased sweating, but more likely to have cough or dyspnea.

Cardiac biomarkers such as creatine kinase and its subfraction CK-MB and troponins are frequently and intermittently increased in patients with renal disease. Thus, investigating acute coronary syndromes in aged patients with chronic kidney disease is very difficult.

2. Chronic kidney disease (CKD)-related bone disease is known as renal or uremic osteodystrophy. It is associated with abnormalities in bone and mineral metabolism that leads to disturbance in calcium, phosphorous, vitamin D, and PTH level.

It consists of a pattern of conditions that are classified based on bone biopsy features including mixed uremic osteodystrophy, osteitis fibrosa, osteomalacia, and adynamic bone disease.

KDIGO has suggested to define CKD-related bone and mineral metabolic disturbances in the setting of a systemic disorder called CKD–mineral bone disease.

Osteoporosis is a condition featured by low bone mass leading to decreased bone strength and increased susceptibility to fractures. Wrist, spine, hip are the common joints involved. Osteoporosis and renal osteodystrophy may occur simultaneously in older patients with CKD, which makes difficult to differentiate.

Evaluation :

Alternate markers of bone metabolism such as serum intact PTH, ionized calcium , phosphorus, alkaline phosphatase, and bicarbonate levels should be measured initially.

Increased parathyroid hormone levels may indicate high turnover bone disease. The desired target level for intact Parathyroid hormone in CKD is not known.

Several diagnostic modalities are available to assess bone health like quantitative computed tomography (qCT), dexa scan and heel ultrasound.

Bone Biopsy remains the investigation of choice for the diagnosis of renal osteodystrophy and evaluation of bone architecture.

The treatment of osteoporosis includes changing modifiable risk factors and using medical management.

(1) Smoking and alcohol intake should be stopped.

(2) Exercise: Moderate physical activity has been related with improvement in bone strength and reduced risk of fractures.

(3) Calcium and vitamin D requirements: Vitamin D deficiency is common in aged individuals commonly during winter. It may be caused

by decreased synthesis, insufficient food intake, dietary restrictions, and sedentary lifestyle in dialysis patients.

The effects of vitamin D on other systems are also more commonly seen.

Deficiency of 25(OH)vitamin D may be associated with increased risk of heart diseases in patients with peritoneal dialysis (PD) and higher risk of acute coronary syndromes in men.

About 2000 mg/d of elemental calcium should be taken in CKD stages 3 to 5. A daily calcium intake of 1200 mg and vitamin D intake of 800 to 1000 IU/d for adults 50 and older is suggested by the national osteoporosis foundation.

(4) Bisphosphonates: Bisphosphonates are useful in managing osteoporosis, but their use in CKD Stages 4 and 5 is doubtful.

(5) Calcitonin: Calcitonin adheres to osteoclasts and prevents bone resorption. It has a few side effects and can be given intranasally. It protects bone mass in post transplant patients.

6.Selective estrogen receptor modulators (SERM) :

Raloxifene was associated with a greater increase in spine BMD, decreased vertebral fractures, and minimal effect on nonvertebral fractures. It can be used safely in women with osteoporosis.

(8) Cinacalcet:

It acts by increasing the sensitivity of calcium sensing receptors in the parathyroid gland to calcium, and plays a important part in homeostasis of parathyroid hormone levels. It helps in modifying bone histology, decreasing bone turnover, and reduction in fibrosis in patients with secondary hyperparathyroidism.

(9) Anabolic agents.

Risk factors for AKI in elderly

1. Comorbid conditions.

The incidence of comorbid conditions that increase the risk for AKI dramatically increases with age. Hypertension, diabetes mellitus, athero-sclerosis, and heart failure are all conditions more commonly

encountered in older individuals, and all can directly or indirectly increase the risk for AKI.

Atherosclerosis and hypertension impair the autoregulatory capacity of the kidney to maintain perfusion in the setting of hypotension. Thus, modest decreases in systemic blood pressure may be associated with significant ischemic damage to the kidney.

More directly, these conditions are associated with the development of glomerulosclerosis and CKD as well as the use of medications (such as ACEs and ARBs) that increase the risk of AKI in certain settings. Another comorbid condition more commonly seen in the elderly is prostatic disease, which when severe can lead to urinary tract obstruction.

2.chronic kidney disease

3.Polypharmacy

Elderly patients are often prescribed number of drugs which may lead to AKI.

4. Structural, functional, and hemodynamic changes in the kidney associated with aging.

a. Structural changes

i. Total renal mass declines with age

ii. The number of functioning glomeruli decreases with age

b. Functional changes

i. Variable decline in GFR with age

ii. A change in sodium metabolism leads to reduced ability to concentrate urine, and increases the susceptibility to volume depletion in the elderly.

c. Hemodynamic changes

i. Decreased renal blood flow and decreased blood flow reserve

ii. Increased renal vascular resistance

iii. Impaired renal vasodilatation and exaggerated renal vasoconstriction

iv. Impaired autoregulation

v. Impaired nitric oxide production

5. Cellular and molecular changes in the aging kidney that increase the risk for AKI.

ETIOLOGY OF AKI IN THE ELDERLY

1.**Prerenal**

The causes of AKI in the aged patients span typical causes seen in other populations. AKI in this circumstance is due to reduced renal blood flow from any cause like heart failure or depletion of effective circulating volume. Older patients are more susceptible to volume depletion and dehydration. In part, this is due to impairment in renal concentrating ability but also to the use of diuretics, impaired thirst sensation and, in some cases, restricted ability to access fluids.

2. intra renal:

1. Vasculature. Acute obstruction of the renal vasculature is an uncommon cause of AKI, but can be seen in the elderly patient undergoing a vascular procedure leading to cholesterol embolization.

2. Acute tubular necrosis (ATN).

Acute tubular necrosis is the commonest cause of AKI in the elderly. Nephrotoxic causes include medications such as aminoglycosides, radio- contrast material, and chemotherapeutic agents.

Pigments attributable to hemolysis or rhabdomyolysis are less common offenders. Major causes include sepsis, severe and prolonged volume depletion, and major surgical procedures (most commonly involving the cardiovascular system).

3. Acute interstitial nephritis (AIN).

4. Glomerulonephritis. A rapidly progressive glomerulonephritis could present as AKI, typically with manifestations in other organ systems. Clinicians should consider this possibility when the urinalysis displays dysmorphic red blood cells, red blood cell casts, and proteinuria. In the elderly, the most common cause of this presentation would be antineutrophil cytoplasmic antibody (ANCA)-associated diseases, most commonly p-ANCA or positive antimyelo- peroxidase.

Postrenal causes of AKI are more commonly encountered in elderly patients. Common causes in the male patient include prostate disease (benign prostatic hypertrophy or prostate carcinoma) or urethral stricture, whereas in females pelvic malignancies are the primary cause.

Kidney Disease Improving Global Outcomes (KDIGO) criteria for the diagnosis of acute kidney injury

Stage 1 AKI:

1.5–1.9 times baseline, OR >0.3 mg/dL rise in the s. creatinine, or urine output <0.5 mL/kg per hour for 6–12 hours

Stage 2 AKI:

2–2.9 times baseline rise in the serum creatinine OR urine output <0.5 mL/ kg per hour for more than 12 hours

Stage 3 AKI:

3 times baseline rise in the s.creatinine, or rise in s.creatinineto more than 4 mg/dL, or urine output less than 0.3 mL/kg per hour for

more than 24 hours, or nil urine output for more than 12 hours, OR the initiation of dialysis, OR, in patients less than 18 years, reduction in glomerular filtration rate less than 35 mL/min per 1.73 m^2

Treatment of AKI

There are no specific therapies for AKI once it is has occurred. Thus the management of AKI is largely supportive, through maintenance of adequate renal blood flow, avoidance of further injury, and renal replacement support.

The decision to initiate renal replacement therapy (RRT) in elderly persons may be difficult and complex, given the possibility that older persons may not fare well on this aggressive, life-sustaining type of therapy and may have comorbid conditions that lead to a very poor overall prognosis.

Glomerular diseases in elderly:

1. Acute nephritic syndrome (ANS) is characterized by the sudden onset of proteinuria and hematuria, often accompanied by hypertension, edema and reduced renal function. An antecedent streptococcal (throat or skin) or an active staphylococcal (skin or soft tissue) infection may be documented (poststreptococcal glomerulonephritis or staphylococcal-related glomerulonephritis).

2. Rapidly progressive glomerulonephritis presents in a more insidious fashion and also manifests hematuria and proteinuria, but is characterized by a progressive and relentless loss of renal function and less evidence of edema and hypertension. Development to end-stage renal disease (ESRD) may occur within few days or weeks. Although an infection may also be present, such features are usually absent, especially in the elderly. If caused by a systemic disease (such as vasculitis) extrarenal symptoms and signs are commonly present (eg, palpable purpura, lung hemorrhage).

3. Nephrotic syndrome is characterized by the abrupt or insidious onset of marked proteinuria (usually more than 3.5 g/d up to more than 20 g/d), with or without hematuria, but with a prominent tendency for edema, hypoalbuminemia, and hyperlipidemia. Renal function is variable and may be normal or reduced.

   In the elderly, the peripheral edema may be severe if there is concomitant congestive heart failure or venous insufficiency.

4. Asymptomatic hematuria and/or proteinuria is characterized by symptom-free (eg, no lower urinary tract symptoms such as dysuria or frequency) excretion of abnormal numbers of intact erythrocytes and/or increased amounts of protein (albumin), or both, in association with normal renal function, normal blood pressure, and absence of edema.

   This presentation is often discovered incidentally during a routine urinalysis, although at times the hematuria may be episodic and gross or macroscopic, thus bringing the patient to the physician's attention. Most often the hematuria is microscopic and persistent or episodic.

5. Chronic glomerular disease can be said to be present when any of the conditions causing glomerular injury have progressed slowly to definitely impaired renal function nearly always with an increased serum creatinine concentration.

One feature of the hematuria observed in the glomerular disease syndromes is the excretion of increased numbers of abnormally shaped, smaller than normal (microcytic), poorly hemoglobinized (hypochromic) erythrocytes in the urine. This is also known as dysmorphic or glomerular hematuria.

Five lesions account for more than 80% of the lesions of primary glomerular disease in the elderly: membranous nephropathy (MN); mesangial, endocapillary, or focal proliferative glomerulonephritis; focal and segmental glomerulosclerosis (FSGS); crescentic glomerulonephritis; and minimal change disease (MCD).

Idiopathic MN is the most commonly encountered glomerular lesion in the elderly with primary glomerular disease . Membranous

nephropathy is characterized by glomerular capillary wall abnormalities with subepithelial deposits of immune complexes that appear electron dense by ultrastructural analysis.

Most patients with MN present with characteristic features of the nephrotic syndrome(>85%), but a few present with asymptomatic proteinuria, often without hematuria. Persistent nephrotic-range proteinuria, particularly more than about 4 g/d, can be associated with a slowly progressive course to ESRD over many years.

There is also a marked susceptibility to thromboembolic diseases, including deep venous thrombosis of the legs, and renal vein thrombosis, sometimes with pulmonary embolism.

Management :

Conservative management (salt restriction, loop diuretics, antihypertensive agents, mainly angiotensin converting enzyme inhibitors [ACEi], angiotensin receptor blockers [ARB], or direct renin inhibitors is indicated when proteinuria is modest(<4 g/d), the symptoms of nephrotic syndrome are tolerable and manageable.

Concomitant therapy with a statin to help reduce the hypercholesterolemia is often indicated. Prophylactic anticoagulants may be indicated when the serum albumin level is low(<2.8 g/dL), if the patient has had a prior thromboembolic event, or has another thrombophilic disorder and is at low risk of a bleeding complication of anticoagulation. Patients with marked proteinuria (>4 g/d for 6 months or longer), those with severe symptomatic nephrotic syndrome, or those with progressive renal disease are candidates for specific therapy.

Cyclical oral cyclophosphamide and glucocorticoids for 6 months (Ponticelli regimen) are preferred for initial therapy.

As an alternative, treatment may be initiated with a calcineurin inhibitor (CNI; cyclosporine or tacrolimus), alone as monotherapy or combined with low-dose steroids for 4 to 6 months, providing that the initial renal function is normal or near normal. Intravenous courses of rituximab, and mycophenolate mofetil (MMF) plus steroids can be used.

Mesangial, endocapillary, or focal proliferative glomerulonephritis is most commonly a manifestation of an underlying streptococcal, staphylococcal, or viral infection. Poststreptococcal glomerulonephritis is not a benign disease in the elderly. It may induce acute congestive heart failure.

Treatment is mainly supportive with diuretics, bed rest, and salt restriction. Staphylococcal-related glomerulonephritismay be associated with IgA deposits in the glomeruli and has a poor prognosis.

IgA nephropathy is common in the elderly . The standard therapy for IgA nephropathy is maximum dosage of an ACEi/ARB or DRI. Combination of an ACEi and an ARB or a DRI may be more effective in lowering protein excretion over the short term, but the long-term benefits and risk of such combination therapy are unknown and side effects may be increased in the elderly with underlying cardiovascular disease.

Patients with proteinuria persisting at more than 1 g/d after 6 months of optimal angiotensin II inhibition therapy and Scr less than 1.5 mg/dl could be treated with cyclical oral and intravenous prednisone.

FSGS is a common lesion found to underlie proteinuria and the nephrotic syndrome in the geriatric population. The characteristic feature of the disease is segmental sclerosis affecting only some glomeruli. IgM and C3 deposits are commonly seen in the sclerosed segments by immunofluorescence microscopy, and ultrastructural analysis shows diffuse effacement of the foot processes.

Patients with the FSGS lesion tend to have an insidious onset of nephrotic syndrome, often present with some degree of renal impairment, and frequently have hypertension. This lesion tends to be uncommon in the very elderly (>80 years of age). Proteinuria may be marked and progressive renal failure is common if marked proteinuria persists. Patients with nonnephrotic proteinuria (< 2 g/d) largely have a benign evolution and can be managed conservatively with diuretics and ACEi, ARB, or DRI.

Crescentic glomerulonephritis is one of the more common primary glomerular lesions found in elderly subjects .This lesion is characterized by abundant multiplication of the cells lining the Bowman space and glomerular podocytes(forming crescents) and by necrotizing lesions in the glomerular capillaries.

Three distinct pathogenetic mechanisms are operative:

(1) antiglomerular basement membrane (GBM) antibodies producing a linear deposit of IgG along the capillary walls; (2) immune complex deposits producing a granular pattern of immunoglobulin along the capillary walls and mesangium, and (3) pauci-immune lesions with relative absence of immunoglobulin in glomeruli.

Pauci-immune lesions are frequently (>80%) associated with circulating antineutrophil cytoplasmic autoantibodies (ANCA) directed to myeloperoxidase or proteinase 3 in the azurophilic granules of leukocytes. The linear deposit of IgG is commonly caused by anti-GBM autoantibody production, whereas the immune complexlesions are commonly caused by lupus nephritis, IgA nephropathy, or infections. These lesions are frequently associated with the clinical pattern of rapidly progressive glomerulonephritis.

The diagnosis of crescentic glomerulonephritis should always be suspected when hematuria and proteinuria are found and the serum creatinine is increasing rapidly. Urgent specific diagnosis of the type of glomerulonephritis by serology (anti-GBM and ANCA in particular) and/ or renal biopsy is indicated.

Minimal change disease:

In younger persons, the lesions are mainly seen at the level of the electron microscope, at which diffuse foot process effacement is evident. Light microscopy is normal or shows only mild mesangial changes and immunofluorescence microscopy is negative or reveals only wisps of IgM in the mesangium. In the elderly, superimposed lesions of arteriolo nephrosclerosis and global glomerulosclerosis may result in imprecision when rendering diagnosis of MCD (by light microscopy alone).

MCD is one of the common cause of the idiopathic nephrotic syndrome in the older adult, accounting for about 10% to 20% of all cases, diminishing in frequency with even more advancing age. MCD in the older adult is typically associated with the abrupt onset of a full-blown nephrotic syndrome with scant hematuria and normal blood pressure for the person's age.

MCD in the elderly is commonly associated with acute kidney injury (AKI). If the nephrotic syndrome is severe, treatment with oral

glucocorticoids, given once daily or every other day in doses of 1 mg/kg/d (maximum dose of 80 mg daily or 120 mg every other day) are usually given.

Adjunctive use of short-term oral cyclophosphamide (1.5–2mg/kg/d for 8–10 weeks) or a CNI-based regimen can be offered for those few patients with frequent recurrences. Treatment must be prolonged in many cases, for 16 to 20 weeks.

Common secondary glomerular diseases in the elderly:

1. Diabetic glomerulosclerosis (diffuse and nodular types) type 2 diabetes

2. Systemic necrotizing and crescentic glomerulonephritis

3. Systemic amyloidosis (AL type, primary and secondary to multiple myeloma)

4. Nonamyloid monoclonal immunoglobulin deposition diseases

5. Idiopathic nodular glomerulosclerosis

6. Malignancy-related glomerulopathy.

7. Drug-related glomerular disease (nonsteroidal anti inflammatory agents, cancer chemotherapeutic agents, bisphosphonates, interferons).

Amyloidosis is a cause of the nephrotic syndrome in about 10% to 15% of elderly patients. It may present as a renal disease without any extrarenal manifestations but more frequently some clues to the

correct diagnosis are present, such as carpal tunnel syndrome, macroglossia, easy bruising, postural hypotension, diarrhea, organomegaly, cardiac disease, or liver disease. In the elderly, AL amyloidosis is the most common disease, but rarely AA amyloidosis or even hereditary amyloidosis may be the cause.

The Congo red stain is positive and 10-nm to 12-nm nonbranching fibrils are found on electron microscopy. Marked proteinuria (up to 20 g/d or more) and some impairment of renal function with mild hypertension is the rule. Plasma levels of free monoclonal immunoglobulin light chains (typically lambda) are frequently increased and a monoclonal paraprotein can often be found in the urine.

About 10% of patients with AL amyloidosis have a frank multiple myeloma on bone marrow examination or bone survey. Nephrotic syndrome and reduced renal function are ominous prognostic signs and, in the absence of therapy, nearly all patients die or progress to ESRD in a matter of a few years from diagnosis . Management include high-dose melphalan plus steroids, or bortezomib and lenalidomide.

Nonamyloid monoclonal immunoglobulin deposition diseases (MIDD) are also a common cause of renal disease in the older population. They may collectively account for 10% to 15% of cases. The diseases consist of light chain MIDD (usually kappa light chain), heavy chain MIDD, light-heavy chain MIDD, IgG MIDD, monoclonal cryoglobulinemia, crystal cryoglobulinemia, and immunotactoid glomerulopathy.

These diseases share in common the deposition of a nonamyloid monoclonal protein in the glomerular capillaries, lacking the β pleated sheet conformation, causing structural and functional deficits including nephrotic syndrome and progressive renal failure.

Idiopathic (nondiabetic) nodular glomerulosclerosis is a recently described condition in which the pathologic findings of nodular DGS are found in patients who have no evidence of diabetes mellitus. The patients tend to be elderly women with a strong history of smoking and hypertension. The pathogenesis is unknown and the prognosis is poor. No effective treatment is available, other than control of blood pressure and stopping smoking.

Goodpasture disease is an uncommon disorder characterized by crescentic glomerulonephritis and circulating antibodies to GBM, often accompanied by diffuse alveolar hemorrhage. In the elderly it is chiefly found in women and overt (alveolar hemorrhage can be observed in less than 50% of cases). There may be a coexisting ANCA-associated nephritis/vasculitis in as many as 20% of cases. With extensive crescents, the prognosis for recovery is poor without aggressive therapy.

Treatment consists of oral cyclophosphamide, oral and intravenous glucocorticoids, and aggressive plasma exchange.

Maintenance immunosuppressive therapy is usually not required unless concomitant ANCA-associated disease is present. Dialysis-dependent patients with serum creatinine greater than 6 mg/dL have only a 10% chance or less of recovery of renal function. Life threatening pulmonary hemorrhage is best treated by high-dose steroids and intensive plasma exchange, which may be life saving.

Systemic pauci-immune necrotizing and crescentic polyangiitis is among the most common secondary glomerular diseases affecting the elderly. This condition is similar to the renal-limited form. The systemic forms are (1) granulomatous polyangiitis (formerly called Wegener granulomatosis) affecting the kidneys, lungs, upper airways, sinuses, sclera, and auditory canal with evident granulomas in the affected vascular tissue; and (2) microscopic polyangiitis (MPA), also affecting the kidneys, lungs, skin, and joints, but without vascular tissue granulomas.

Both are strongly associated (>90%) with ANCA; mostly antiproteinase 3 in GPA and anti myeloperoxidase in MPA. Systemic features of fever, cough, myalgias, Pulmonary hemorrhage, necrotizing cutaneous angiitis, sinusitis, and upper and lower airway disease can be present.

The treatment is identical to that described earlier for renal limited necrotizing and crescentic glomerulonephritis but the prognosis may be poorer because of the multiorgan involvement. Patients who are dialysis dependent for less than 2 weeks are likely to benefit from the

addition of intensive plasma exchange but the risk of complications is great. Courses of rituximab are emerging as an alternative to cyclophosphamide-glucocorticoid regimens, especially in relapsing disease.

A high degree of suspicion for underlying glomerular disease needs to be present when hematuria and proteinuria are present concomitantly and an increasing serum creatinine should be viewed with a sense of urgency.

The atypical clinical features of acute and chronic glomerular disease in the elderly should be remembered.

Vascular diseases in elderly:

Causes of vascular injury to kidney:

When classified by size of the vessel, the vascular diseases can be classified into those affecting (1) the microscopic vessels, (2) arterioles, and (3) the main renal arteries.

A rise in renal vascular resistance and decline in effective renal blood flow are observed as BP decreases. Although the GFR is relatively maintained, renal insufficiency may occur in some hypertensive patients.

As age advances, the total number of effective glomeruli decrease by 30 to 50% as the number of sclerotic or abnormal glomeruli increases. The ischemic changes are modifiable initially.

However, with hyalinosis, the pathology continues to glomerular sclerosis. With continued pathogenesis the lesion progresses to the formation of aglomerular arterioles. When the vascular lesions involve most of the glomeruli and severe, the entire kidney undergoes ischemic atrophy.

Intrarenal Arterioles and Interlobular Arteriolar Disease:

Hypertension, arteriosclerosis and aging affect the arterioles in the kidney causing afferent and efferent arteriolar atrophy. Impairment of auto regulation with glomerular vasodilatation occur.

This is accompanied by hypertrophy of the intact nephrons leading to intraglomerular hypertension, excretion of albumin in urine, and sclerosis of glomeruli. The histologic changes like hyperplastic elastic arteriosclerosis, intimal thickening, reduplication of the lamina elastica interna, and hyalinization are seen. These changes lead to decline in renal blood flow along with progressive ischemic changes seen in the glomeruli and renal tubules.

Atherosclerotic Renal Artery Stenosis

It is the presence of cross-sectional arterial luminal constriction, which may or may not have any important hemodynamic effects. Atherosclerotic renal artery stenosis commonly causes occlusion of the ostium and the early one third of the renal arteries.

Only when the luminal diameter is reduced by 70% atherosclerotic RAS often becomes clinically significant. Clinically severe stenosis leads to parenchymal ischemia, atrophy, and loss of kidney function.

Diagnosis of atherosclerotic RAS :

There is no pathognomonic clinical signs. . The development of hypertension after 50 yrs of age, presence of abdominal bruit, atrophic kidney, frequent pulmonary edema or the occurrence of AKI after initiation of ACE inhibitors(ACEi) or ARBs may suggest the presence of renal artery stenosis.

Imaging modalities used are Doppler ultrasound, MR angiography with Intravenous gadolinium, CT Angiogram, renal artery angiography(gold standard), nuclear renal scan.

Treatment :

All cases of RAS need not be treated because some patients will have no clinical symptoms. However, RAS may cause ischemic nephropathy with progressive CKD or it may cause hypertension.

Percutaneous transluminal renal angioplasty (PTRA) with stents has different results depending on the patient selection

Urinary tract infections in the elderly :

Asymptomatic bacteriuria (ASB) is a common manifestation in elderly people. The ASB is diagnosed based on a urine culture from a urine specimen that has minimal contamination from a person without symptoms or signs referable to urinary infection. For asymptomatic female, bacteriuria is defined as 2 successive voided urine specimens with growth of the same bacterial species in quantitative counts $>10^5$ colony forming units (cfu)/ml. For asymptomatic male, bacteriuria is defined as a single, clean-catch voided urine specimen with single bacterial strain grown in a quantitative count 10^5 cfu/ml.

Selection and management of ASB in aged persons is only recommended in the following two situations:

(1)Before performing TURP and (2) before urologic surgeries in which bleeding from the mucosa is expected.

Old people with various diseases may not be able to report their symptoms. The only clinical symptoms that have been present with

bacteriuria plus pyuria in hospitalized adults with clinically suspected UTIs are dysuria, altered mental status, and abnormal urine analysis.

Empirical therapy with a third-generation cephalosporin is suitable drug therapy for older adults admitted with urosepsis until culture and susceptibility reports are available. Nitrofurantoin, trimethoprim-sulfamethoxazole, and fluoroquinolones are suitable first-line drugs for outpatients.

I. for individuals who do not have an indwelling catheter, minimum criteria for starting antibiotics include acute dysuria alone or fever (37.9°C)

   and at least one of the following: urgency, frequency, suprapubic pain, gross hematuria, costovertebral angle tenderness, or urinary incontinence.

II. For residents who have a chronic indwelling catheter, minimum criteria for initiating antibiotics include the presence of at least one of the following: fever, rigors, new costovertebral angle tenderness ,or new onset of confusion.

One of the most predominant urologic problems in elderly persons, is lower urinary tract symptoms (LUTS).Benign prostatic hyperplasiais a disorder that affects the most of aged patients.

LUTS are evaluated with both subjective and objective tests. The American Urological Association BPH symptom score was designed to evaluate subjective complaints. Patients with lower scores are given life style changes, with intermediate scores are given the choice of medical management and patients with high scores are given the choice of transurethral surgery.

Patients with hydronephrosis, hematuria, acute retention of urine and recurrent infection are advised surgery. Cystoscopy and urodynamic studies are the investigations of choice. Surgical management include laser ablation, resection and microwave or radiofrequency therapy.

Medical management:

Alpha 1 blockers relaxes the smooth muscle fibers of the prostatic gland and greatly improve urine flow. Common nonselective alpha 1 blockers are terazosin and doxazosin; selective agents are tamsulosin and alfuzosin.

5 alpha reductase inhibitors prevent the conversion of testosterone to dihydro testosterone, which is a potent inducer of prostatic glandular tissue. This decline in local androgen stimulation results in a reduction of prostatic volume .

Prostate cancer: About 10% of patients above 65 yr of age have been found to have prostate cancer. The American Urologic Association suggests screening with digital rectal exam and PSA testing annually, beginning at age 50. A PSA level between 4 and 10 ng/ml represents a 25% risk of prostate cancer. Patients detected to have an abnormal DRE or an increased PSA are the candidates for prostate biopsy.

Polypharmacy :

An aged patient is on many prescribed medications, as well as over-the-counter medications. Many of these drugs can have urologic adverse effects. Many common drugs, used for common cold and upper respiratory infection have anti-cholinergic properties that can increase LUTS. The drug list of all aged patients must be analysed to confirm that lower urinary tract signs or symptoms are not drug related.

MATERIALS AND METHODS

PLACE OF STUDY:

DEPARTMENT OF NEPHROLOGY, NEPHRO OPD AND WARDS, AND MEDICAL WARDS AT STANLEY MEDICAL COLLEGE AND HOSPITAL, CHENNAI.

DURATION:

June 2014 TO SEP 2014.

STUDY DESIGN:

PROSPECTIVE OBSERVATIONAL STUDY

SAMPLE SIZE: 75

ETHICAL COMMITTEE APPROVAL:

Study was conducted only after getting approval of the institutional ethics committee. A copy of the approval is enclosed .

SELECTION OF PATIENTS:

INCLUSION CRITERIA

1. Patients age more than 65 years

2. Patients with raised urea, creatinine values

3. Patients with hematuria, proteinuria, abnormal urine sediments

4. Patients with electrolyte imbalance

EXCLUSION CRITERIA

Patients less than 65 years.

METHODOLOGY:

Elderly patients above 65 years with raised creatinine and abnormal urinalysis reports coming to OPD/admission unit/wards from June 2014 to September 2014 are included in the study. Patients will be subjected to symptom analysis, clinical examination, laboratory investigations and Imaging studies. The final analysis will be made at the end of the study to achieve the fore mentioned goals using appropriate statistical methods.

RESULTS AND DISCUSSION

This study included 75 patients. Patients were subjected to urine analysis, blood investigations, USG abdomen and other investigations as needed as per the proforma. Renal biopsy was done in 6 subjects as it was required for diagnosis .8 patients died during the study due to various reasons.

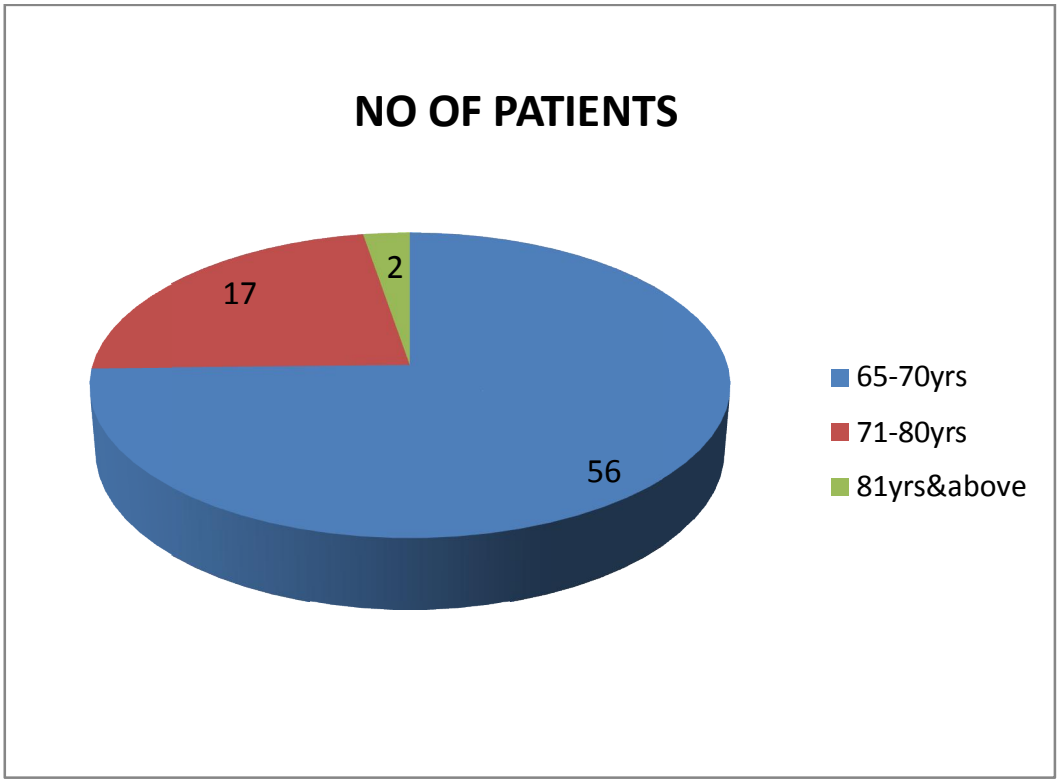
ANALYSIS OF DATA OF STUDY GROUP:

AGE DISTRIBUTION:

Age (years)	Number of patients	Percentage
65-7O	56	74.66
71- 80	17	22.66
>80	2	2.66

TABLE 1: AGE DISTRIBUTION OF STUDY GROUP

Among the 75 patients studied most of the patients were between 65 and 70 years. 56 (76.66%) patients were between 65-70 years, 17 patients were between 71 - 80 years. Only 2 (2.66%) patients were above 80 years

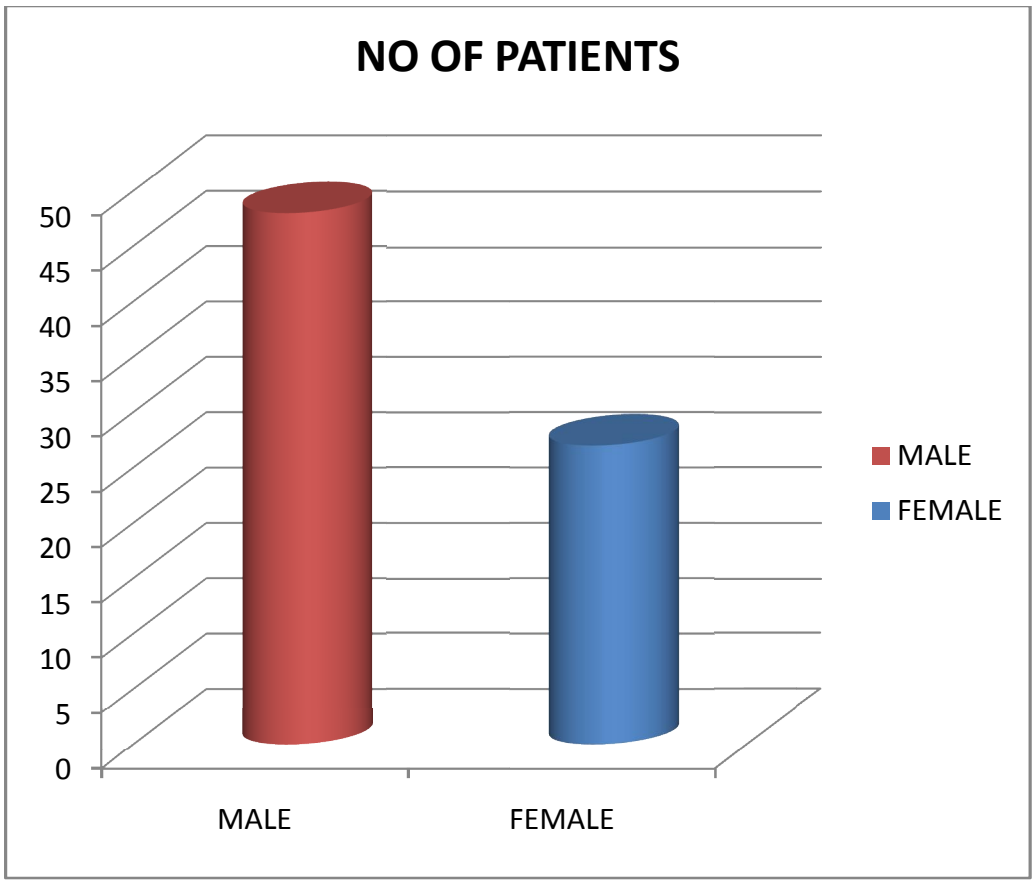


SEX DISTRIBUTION OF STUDY GROUP:

GENDER	NO.OF PATIENTS	PERCENTAGE
MALE	48	64
FEMALE	27	36

TABLE 2: SEX DISTRIBUTION OF STUDY GROUP

Among the 75 patients, 48 (64%) were males and 27(36%) patients were females.



PREVALENCE OF DIABETES AND HYPERTENSION IN STUDY GROUP:

	No. of patients	Percentage
DIABETES	**39**	**52**
HYPERTENSION	**26**	**34.66**
BOTH	**7**	**9.33**

TABLE 3 : Prevalence of Diabetes and Hypertension in Study Group

The prevalence of diabetes in the study group was 39 patients (52%)and that of hypertension was among 26 (34.66%) patients. Both diabetes and hypertension were seen in 7 (9.33%)patients.

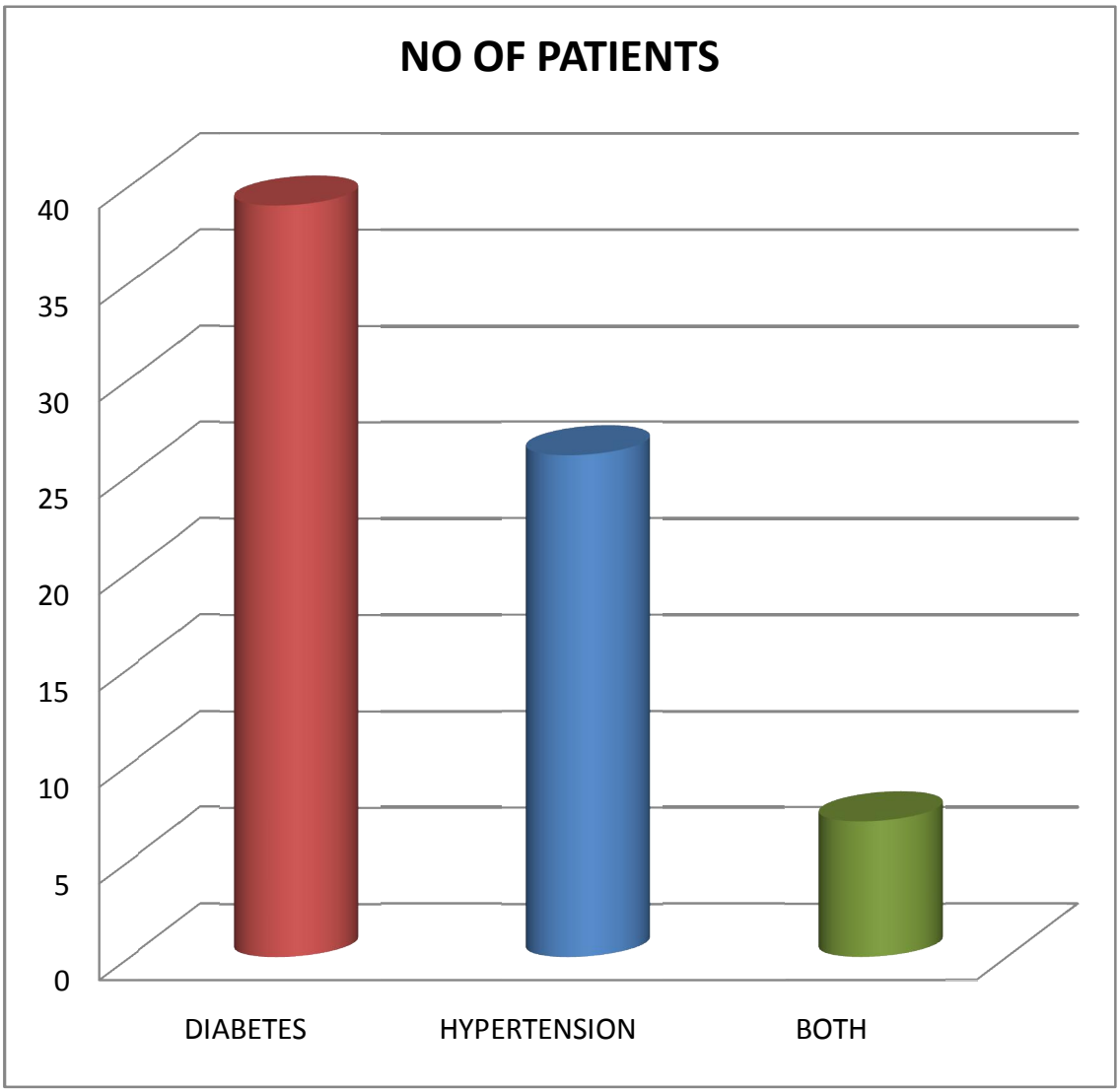
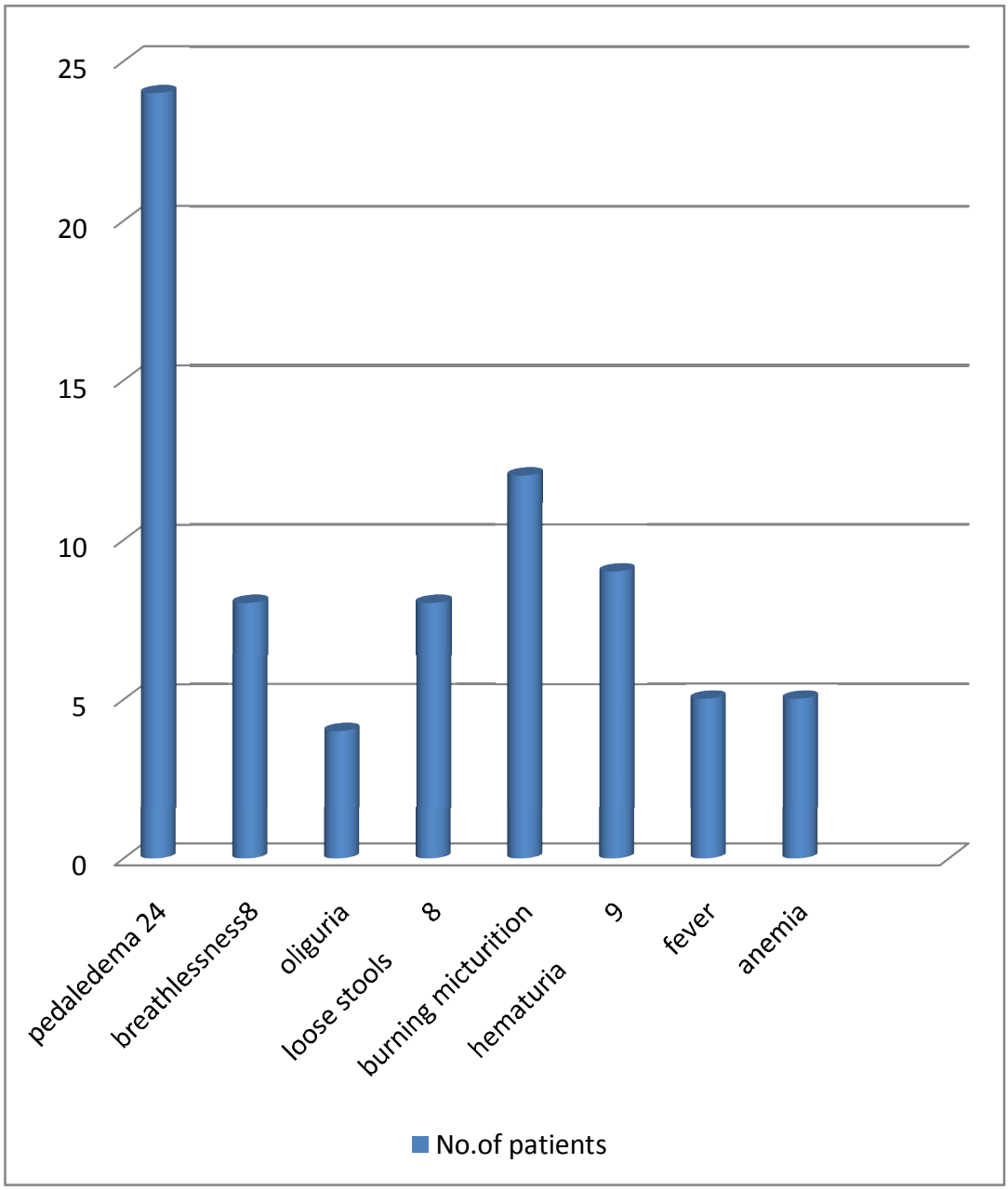

NO OF PATIENTS
40
35
30
25
20
15
10
5
0
DIABETES
HYPERTENSION
BOTH

TABLE 4: DISTRIBUTION OF SYMPTOMS IN THE STUDY POPULATION

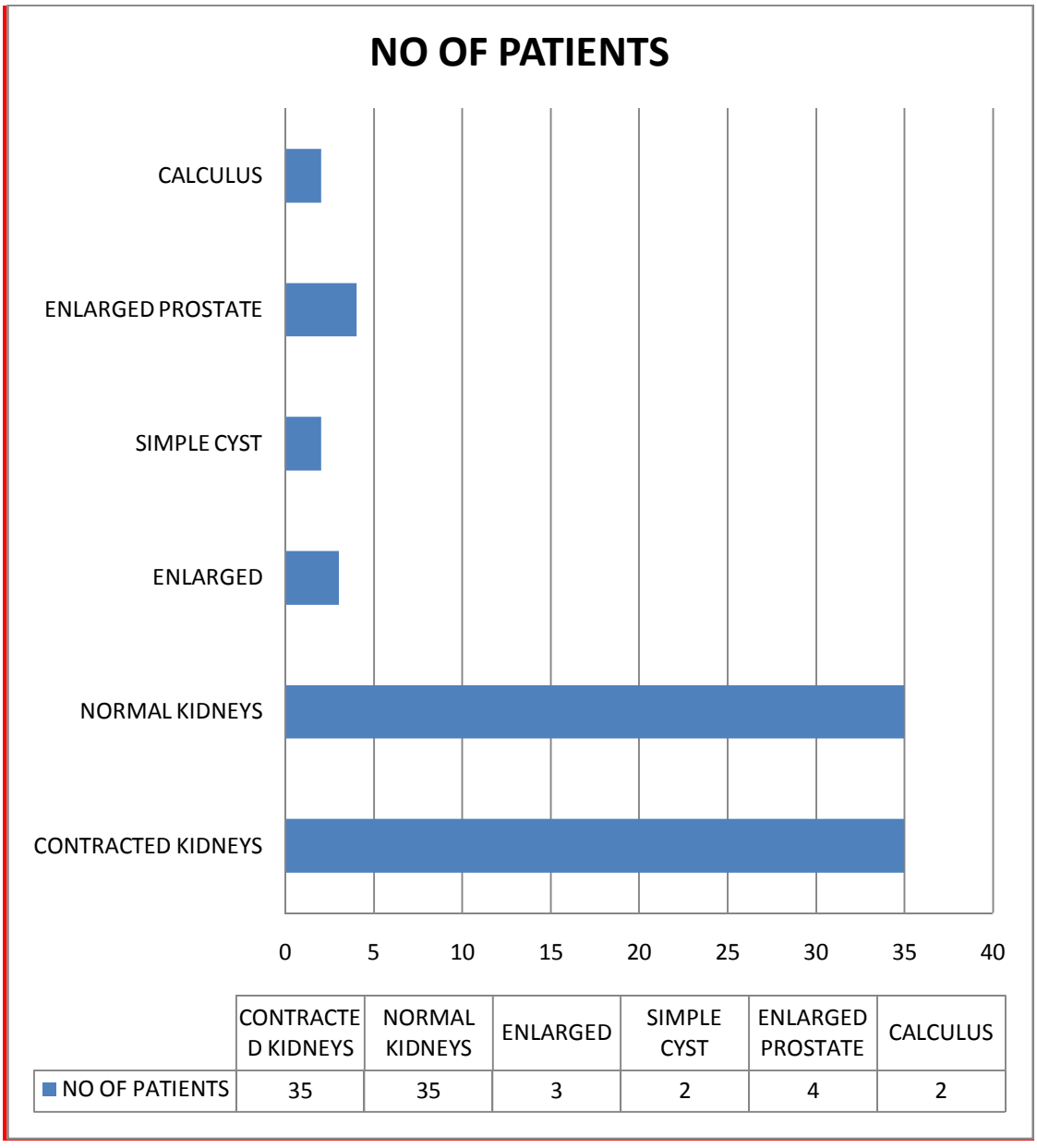
SYMPTOMS	NO.OF PATIENTS	PERCENTAGE
Pedal edema	24	32
breathlessness	8	10.66
oliguria	4	5.33
Burning micturition	12	16
hematuria	9	12
fever	5	6.66
anemia	5	6.66


25
20
15
10
5
0
pedaledema 24
breathlessness8
oliguria
loose stools
8
burning micturition
hematuria
9
fever
anemia
No.of patients

ULTRASOUND FINDINGS IN THE STUDY GROUP:

USG FINDINGS	NO.OF PATIENTS	PERCENTAGE
Contracted kidneys	35	46.66
Normal sized kidneys	35	46.66
Enlarged kidneys	3	4
Simple cysts	2	2.66
Enlarged prostate	4	5.33
Renal/ureteric calculi	2	2.66

TABLE 5 : DISTRIBUTION OF USG FINDINGS IN STUDY GROUP



	CONTRACTED KIDNEYS	NORMAL KIDNEYS	ENLARGED	SIMPLE CYST	ENLARGED PROSTATE	CALCULUS
NO OF PATIENTS	35	35	3	2	4	2

The USG findings in the study group was as follows : contracted kidneys found in 35(46.66%) patients which indicates end stage renal disease(ESRD). Normal sized kidneys found among 35(46.66%) patients. Increased size with multiple cysts seen among 3(4%) patients. Simple cysts seen among 2(2.66%) patients. Enlarged prostate seen in 4 (5.33%) patients. Renal /ureteric calculi seen in 2 (2.66%) patients

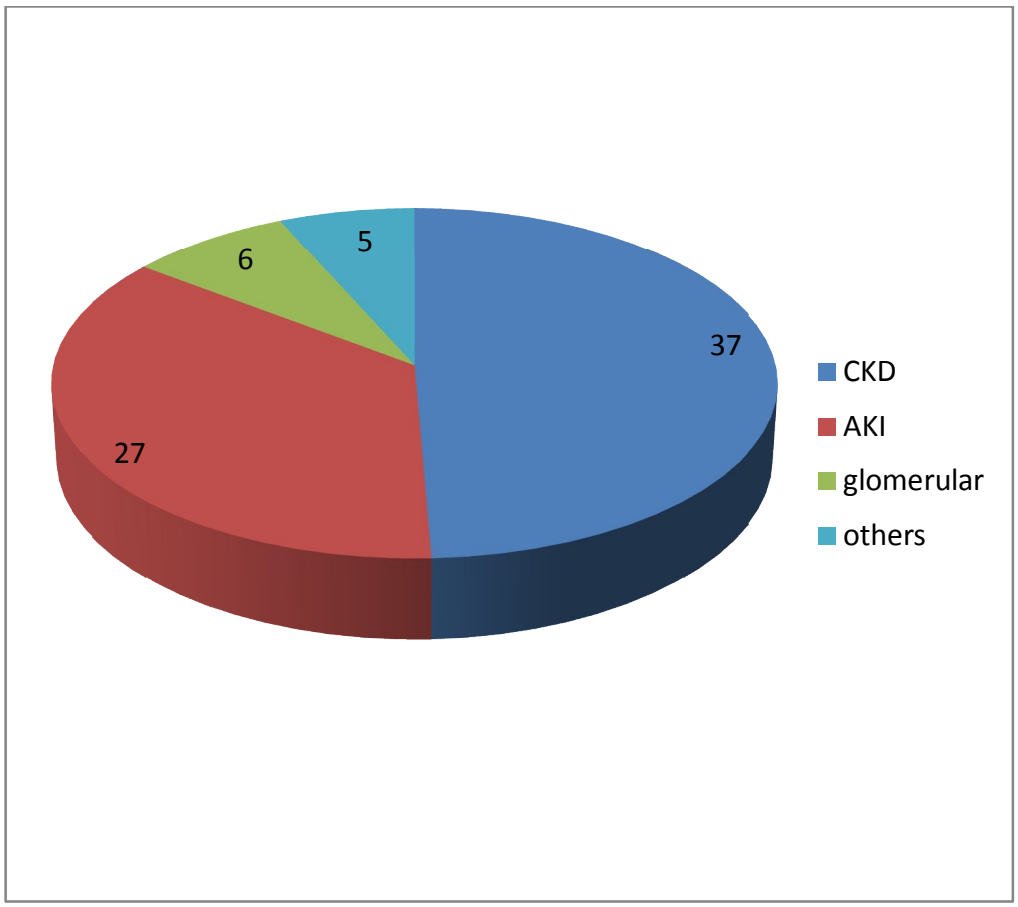
PATTERN OF RENAL DISEASES IN THE STUDY POPULATION :

DIAGNOSIS	NO.OF PATIENTS	PERCENTAGE
CKD	37	49.33
AKI	27	36
Glomerular diseases	6	8
Others	5	6.66

TABLE 6 : PATTERN OF RENAL DISEASES IN THE STUDY POPULATION

Among the 75 patients, 37 patients(49.33%) were found to have CKD. 27 patients (36%) had AKI. Glomerular diseases seen in 6 patients (8%). This included membranous nephropathy among 2 patients, post infectious glomerulonephritis (2 patients), myeloma

kidney (2 patients). Other diseases contributed to 6.66% of the study population. This includes polycystic cystic kidney among 3 patients, simple cysts seen in 2 patients.

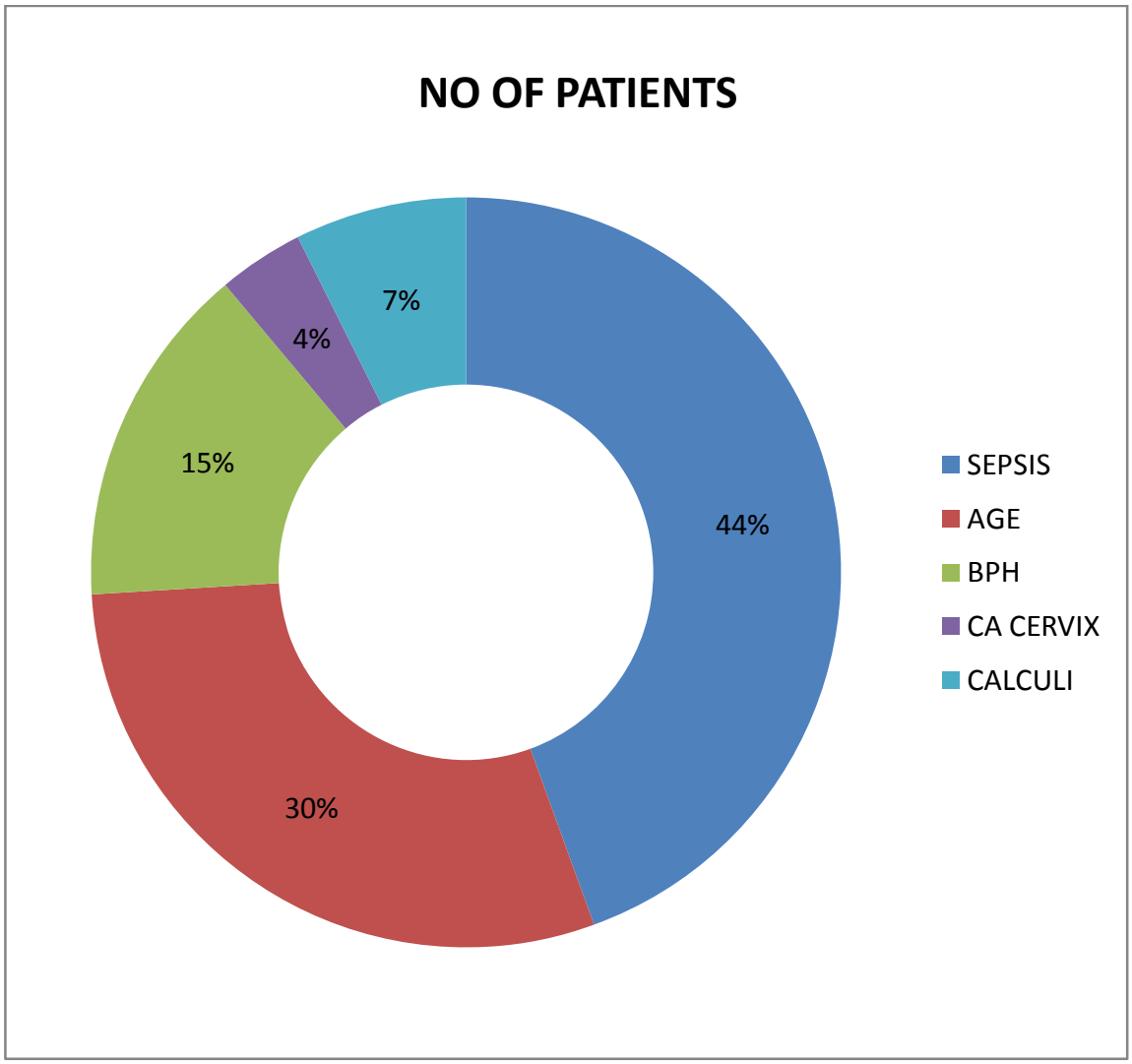


ETIOLOGY OF AKI IN THE STUDY POPULATION:

Causes	No. of Patients	Percentage
sepsis	12	44.44
Acute gastroenteritis	8	29.62
BPH	4	14.81
Ca cervix	1	3.70

Among the study population, 27 patients (36%) had acute kidney injury(AKI).

Sepsis contributed to AKI in 12 patients (44.44%). Acute gastroenteritis leading to dehydration was seen in 8 patients(29.62%). Others were BPH in 14.81%, carcinoma cervix in 3.70% and calculi in 4.7% patients.


NO OF PATIENTS
44%
30%
15%
4%
7%
SEPSIS
AGE
BPH
CA CERVIX
CALCULI

CITATION

1**. Affifi et al** has done a study among 220 elderly patients in Ain shams university and Nasser institute hospitals, cairo, Egypt. This study showed diabetic nephropathy in 28.2%, hypertensive nephrosclerosis 25.5%, UTI, cystitis and pyelonephritis in 6.8%, renal stones in 5.9%, obstructive nephropathy in 7.6%, simple cysts in 4.5%, CRF of unknown origin in 13.1%. Multiple myeloma, lupus nephritis, vasculitis recorded in few numbers.

2. **Prakash. J, Saxena RK, Sharma OP et** al has done a study among 200 hundred patients over the age of 60 years in india. The clinical presentation included chronic renal failure (42.5%),acute renal failure (28%); nephrotic syndrome (14.5%); acute glomerulonephritis (7.5%); renal vascular disease (5%) and renal cystic disease (2.5%). Diabetic nephropathy, obstructive uropathy and hypertensive nephrosclerosis were the major causes of CRF, accounting for 80% of total CRF in the elderly. Prerenal ARF, obstructive uropathy and sepsis were contributing factors for ARF in 82% of the cases. Volume depletion due to gastrointestinal fluid loss and urinary tract obstruction on account of enlarged prostate were the leading causes of ARF in 20 (35.7%) and 8 (14.3%) cases respectively.

3. **Agarwal et al** has done a study on spectrum of renal diseases in indian adults. Chronic renal failure (CRF), nephrotic syndrome (NS), nephritic syndrome and hypertension were the four common presentations seen in 47.8%, 15.03%, 4.6% and 4.9% cases respectively.

4. **Prakash J et al** has done a study on glomerular diseases in elderly people.

The clinical presentation of GN included: nephrotic syndrome 61.5%, acute nephritic syndrome 29.2%, rapidly progressive GN 6.15% and asymptomatic urinary abnormality 3.%. Overall, primary and secondary glomerular disease were seen in 72.3% and 27.6% elderly patients.

5 .Renal disease in the elderly and the very elderly Japanese: analysis of the Japan Renal Biopsy Registry (J-RBR). Primary glomerular disease was the most frequent diagnosis, followed by MPO-ANCA-positive nephritis, IgA nephropathy (IgAN), and diabetic nephropathy.

CONCLUSIONS

1. The study was done to know about the pattern of renal diseases among elderly patients. Majority of the patients were found to have **CKD**(49.33%).

Diabetes and hypertension are the major risk factors in CKD patients.

2. **AKI** was seen in 36% patients. Sepsis contributed to large part of AKI (44.4%). dehydration due to gastroenteritis contributed to AKI in 29.62% patients. Other causes are BPH in 14.81% and Ca cervix in 3.7% patients.

3. Glomerular diseases were seen in 6 patients (8%). This includes membranous nephropathy (2 patients), acute glomerulonephritis (2 patients) and myeloma kidney seen in 2 patients.

4. Other diseases seen in 6.66% of study population. This includes polycystic kidney seen in 3 patients, simple cysts in 2 patients.

Good control of diabetes and hypertension in younger age may prevent the occurrence of CKD as they grow older. Elderly patients should avoid taking over the counter drugs.

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PROFORMA

PRELIMINARY DATA OF THE PATIENT

Name: Age: Sex: IP NO:

Address: PH No:

DOA: **DOD:**

Occupation: Income: Religion:

Chief complaints

PAST H/O: Diabetes Mellitus / Hypertension/ CKD/ Heart disease/ Syphilis/ TB/ /Seizures/

Surgical history if any :

Drug Intake :

FAMILY HISTORY :

PERSONAL HISTORY“ :

Diet:Veg/Non Veg ;

Smoking :

Alcohol intake:

Bowel and Bladder: Regular/altered

Marital status: married /unmarried

Menstrual history if any: Age of menarche / menopause; Regular /irregular cycles

Treatment history: if any

GENERAL PHYSICAL EXAMINATION

Built & Nourishment: Height in cm; Weight in Kg

Pallor / Cyanosis /Clubbing / Oedema / Icterus / Lymphadenopathy

Carotid Bruit

Evidence of any congenital abnormalities

VITAL PARAMETERS

Pulse:

Peripheral pulses:

BP :inmmHG :

RR: .../min

JVP:

CVS:

RS :

ABDOMEN;

INVESTIGATIONS:

Blood:

Hb% TC, DC ;Bl Urea: S.Creatinine:

RBS/ FBS / PPBS S Electrolytes : Na K

Urine Routine Examination: Albumin Sugar deposits

CXR-PA View:

ECG:

ESR:

Serum CRP:

Serum Uric acid

USG ABDOMEN

ANA,C3 ,C4(if necessary))

RENAL BIOPSY(if necessary)

OTHER INVESTIGATIONS(as required):

PROVISIONAL / CLINICAL DIAGNOSIS

சுயஒப்புதல்படிவம்

அரசு.ஸ்டான்லிமருத்துவகல்லூரி, சென்னை – 600001

மூன்றாம்நிலைசிகிச்சைக்கானமருத்துவமனைக்குவரும்முதியவர்களுக்குஏற்படும்பல்வேறுவகையானசிறுநீரகநோய்கள்பற்றியஒருஆய்வு

ஆய்வாளர்: மருவீரபாண்டியன்.
முதுநிலைபட்டமேற்படிப்புமாணவர்,
பொதுமருத்துவப்பட்டபடிப்பு.
வழிகாட்டி : பேராசிரியர்மரு.எட்வின்பெர்ணான்டோ
பேராசிரியர், சிறுநீரகவியல்துறை
அரசுஸ்டான்லிமருத்துவமனை.

இந்தமருத்துவஆய்வின்விவரங்கள்எனக்குவிளக்கப்பட்டது.என்னுடையசந்தேகங்களைகேட்கவும்,அதற்கானதகுந்தவிளக்கங்களைப்பெறவும்வாய்ப்பளிக்கப்பட்டது.

நான்இவ்வாய்வில்தன்னிச்சையாகத்தான்பங்கேற்கிறேன்.எந்தகாரணத்தினாலும், எந்தகட்டத்திலும்,எந்தசட்டசிக்கலும், இன்றிஇந்தஆய்விலிருந்துவிலகிக்கொள்ளலாம்என்றும்அறிந்துகொண்டேன்.

நான்ஆய்விலிருந்துவிலகிக்கொண்டாலும்ஆய்வாளர்என்னுடையமருத்துவஅறிக்கைகளைப்பார்ப்பதற்கோஅல்லதுஉபயோகிக்கவோஎன்அனுமதிதேவையில்லைஎனவும்அறிந்துகொண்டேன்.என்னைப்பற்றியதகவல்கள்இரகசியமாகப்பாதுகாக்கப்படும்என்பதையும்அறிவேன்.

இந்தஆய்வில்எனக்குஎவ்விதபரிசோதனைகளையும், சிகிச்சைகளையும்மேற்கொள்ளநான்முழுமனதுடன்சம்மதிக்கிறேன்.

இப்படிக்கு

ஆய்வாளர்கையொப்பம் **நோயாளியின்கையொப்பம்**

KEY TO MASTER CHART

RISK FACTORS

DM	diabetes mellitus
HT	Hypertension
AGE	Acute gastroenteritis
BPH	Benign prostate hypertrophy

SYMPTOMS

Pedaledema	1
Breathlessness	2
Oliguria	3
Diarrhoea,vomiting	4
Burning micturition	5
Hematuria	6
Fever	7
Anemia	8

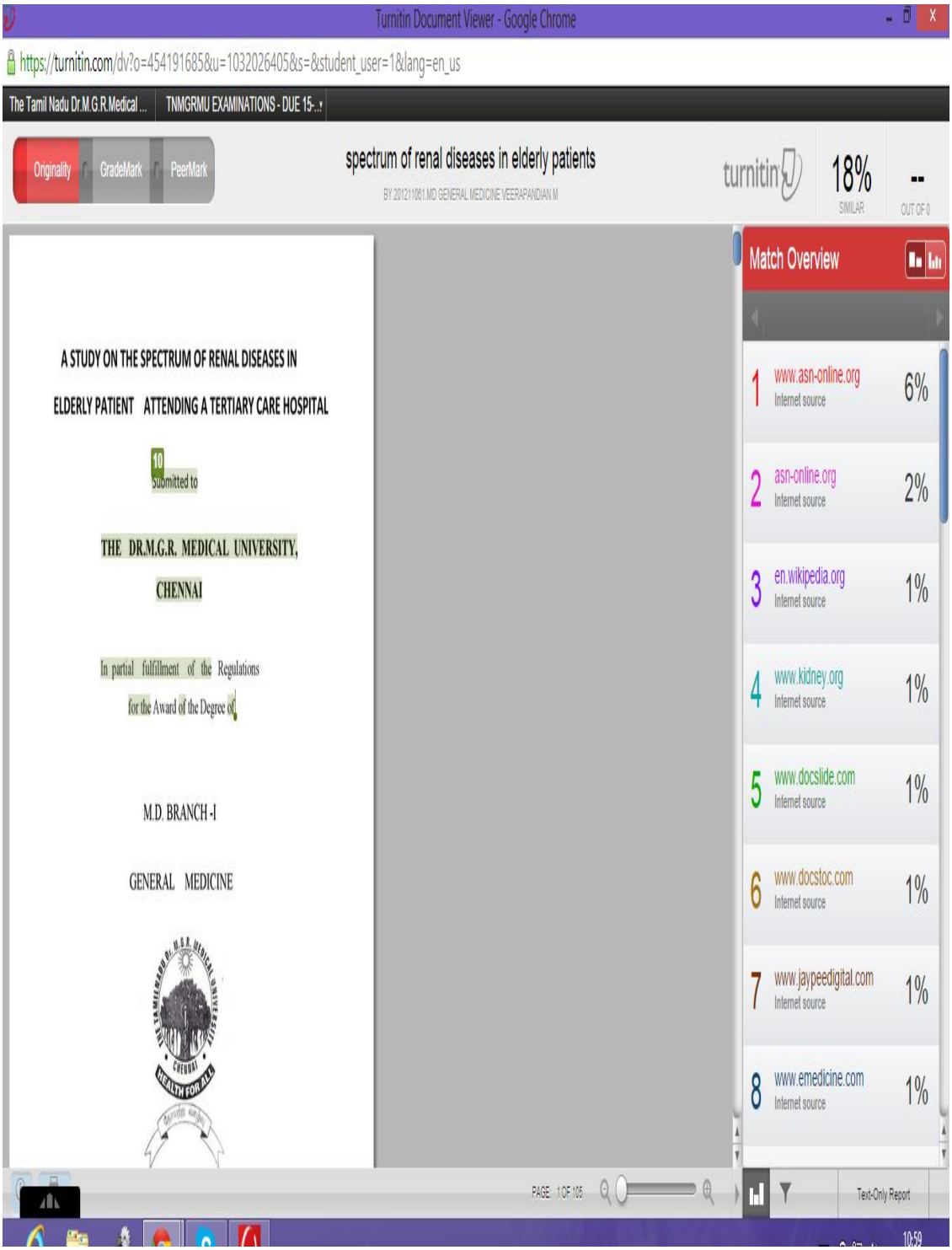
USG FINDINGS

C	Contracted kidneys
N	Normal kidneys

MASTER CHART

S.NO	AGE	SEX	RISK FACTORS	symptoms	UREA	S.CREATININE	URINE ANALYSIS	USG FINDINGS	DIAGNOSIS
1	72	M	DM, HT	1	56	2.4	1+	C	CKD
2	68	M	sepsis,DM	3	68	2.5		N	AKI
3	75	F	DM	2	125	4.2	1+	C	CKD
4	74	M	HT	2	112	3.6	1+	C	CKD
5	65	M	DM,HT	1	96	3.5	1+	C	CKD
6	68	M	HT,BPH	5	62	3.2		N,enlarged prostate	AKI
7	82	F	DM, sepsis	3	56	2.8		N	AKI
8	72	F	AGE	4	84	4.1		N	AKI
9	75	M	DM ,HT	2	156	6.3	2+	C	CKD
10	66	M	DM	1	126	4	1+	C	CKD
11	75	F	HT,sepsis	3	72	2.6		N	AKI
12	68	M	BPH	5	64	2.9		N,enlarged prostate	AKI
13	66	F	DM,sepsis	7	58	2.8		N	AKI
14	73	M	DM	2	162	8.2	2+	C	CKD
15	76	F	AGE	4	54	2.9		N	AKI
16	69	M	AGE ,HT	4	76	2.9		N	AKI
17	74	F	DM	1	170	8.5	1+	C	CKD
18	75	M	BPH	5	84	4.5		N,enlarged prostate	AKI
19	68	M	DM	1	78	4.7	1+	C	CKD
20	77	M	sepsis,HT	7	55	2.5		N	AKI
21	66	M	sepsis,DM	5	112	3.8		N	AKI
22	78	F	HT	1	84	3.9	trace	C	CKD
23	65	M	DM,HT	8	179	9.3	1+	C	CKD
24	68	M	DM	1	102	3.4	1+	C	CKD
25	70	M	HT	5	98	4.5		increased	ADPKD
26	68	M	sepsis,DM	7	58	2.4		N	AKI
27	67	F	HT	1	88	3.3	1+	C	CKD
28	68	F	BPH	5	76	2.9		N,enlarged prostate	AKI
29	74	M	DM	1	132	4.5	2+	C	CKD
30	69	M	HT	5	94	4.6		increased	ADPKD
31	65	F	DM	1	81	4.4	2+	C	CKD
32	66	M	sepsis,HT	3	60	3		N	AKI
33	65	M	DM	2	162	8.8	2+	C	CKD
34	67	M	DM	6	96	4.6	protein 3+	N	myeloma
35	68	F	sepsis	3	53	2.6		N	AKI
36	74	M	AGE, HT	4	76	3.4		N	AKI

37	68	M	HT	1	78	3.2	1+	C	CKD
38	66	F	AGE	4	84	3.1		N	AKI
39	70	M	DM	1	94	4.5	1+	C	CKD
40	68	M	DM	8	124	4.9	1+	C	CKD
41	66	M		6	92	4		simple cysts	simple cysts
42	76	M	sepsis,HT	7	78	3.2		N	AKI
43	68	F	DM,HT	1	142	6.5	1+	C	CKD
44	66	F	ca cervix	5	84	3.5		N	AKI
45	66	F	sepsis, DM	7	53	2.6		N	AKI
46	65	M	HT	1	64	2.5	1+	C	CKD
47	65	M	HT	6	88	3.1		increased	ADPKD
48	71	M	DM,HT	6	84	3.4		simple cysts	simple cysts
49	73	F	AGE	4	58	1.9		N	AKI
50	68	M	DM	1	102	3.5	1+	C	CKD
51	65	F	DM	6	78	4.1	protein 3+	N	myeloma
52	69	M	AGE,DM	4	68	2.4		N	AKI
53	68	M	calculi	5	76	3.6		N, renal calculi	AKI
54	66	M	HT	8	88	4.5	1+	C	CKD
55	68	F	DM	2	156	7.4	2+	C	CKD
56	65	M	HT	1	86	4.3	1+	C	CKD
57	69	F	HT	1	108	5.4	1+	C	CKD
58	68	M	DM	5	70	1.8		N	DN
59	65	F	calculi	5	65	2.3		N,ureteric calculi	AKI
60	66	M	DM,HT	1	180	11	2+	C	CKD
61	65	M	DM	8	126	5.5	1+	C	CKD
62	70	F	DM	6	88	3.4	2+,RBC 1+	N	acute GN
63	66	F	HT	1	86	3.5	1+	C	CKD
64	68	M	DM	6	88	4.5	3+	N	MN
65	66	M	DM	1	103	3.5	1+	C	CKD
66	67	F	DM	6	98	4.3	2+,RBC2+	N	acute GN
67	66	M	HT	6	76	3.6	3+	N	MN
68	65	F	DM	1	66	2.3	1+	C	CKD
69	65	M	HT	1	112	4.5	1+	C	CKD
70	66	F	DM	2	129	4.8	Trace	C	CKD
71	70	M	sepsis	3	58	2.6		N	AKI
72	65	M	HT	8	66	3.2	1+	C	CKD
73	65	F	DM	1	162	8.3	1+	C	CKD
74	68	M	DM	5	56	1.5	2+	N	DN
75	84	M	AGE	4	74	2.8		N	AKI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A STUDY ON THE SPECTRUM OF RENAL DISEASES IN
ELDERLY PATIENT ATTENDING A TERTIARY CARE HOSPITAL
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Submitted to
THE DR.M.G.R. MEDICAL UNIVERSITY,
CHENNAI
In partial fulfilment of the Regulations
for the Award of the Degree of
M.D. BRANCH - I
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Text-Only Report

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A Study on Spectrum of Renal Diseases in elderly patients attending a tertiary care Hospital

Principal Investigator : Dr. Veerapandian.M

Designation : PG in MD (General Medicine)

Department : Department of General Medicine
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 02.07.2014 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI